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THE OSTEOPLASTIC WALL THORACOPLASTY AFTER PNEUMONECTOMIES

VIKING OLOV BJÖRK, M.D.
STOCKHOLM, SWEDEN

THE PRINCIPLE of simultaneous pneumonectomy and thoracoplasty is not new. As early as 1933, when Evarts Graham performed the historic pneumonectomy for carcinoma of the lung, he completed this operation with a resection of ribs three to ten. Graham continued to use simultaneous thoracoplasty in the first 5 or 6 cases of pneumonectomy but then left this practice as a routine. In cases with pneumonectomy for tuberculosis, however, he continued to perform a simultaneous thoracoplasty in order to diminish the possibility of tuberculous empyema. Later Iverson, and Skinner,[6] 1950, Conklin, Tuhy, and Grismer,[4] 1951, and Samson,[7] 1951, advocated thoracoplasty at the same stage as pneumonectomy for tuberculosis.

The name thoracoplasty means to mold the chest. Earlier techniques, however, have mainly consisted of rib resection. A stable chest wall is then only obtained when the rib regeneration has occurred. This new chest wall may have the desired size and shape, but will be greatly influenced by the scapula and the re-expanding lung.

As the name thoracoplasty is generally accepted for any kind of rib resection, the name osteoplastic has been added to the procedure I have devised to diminish and mold the chest into a desired shape. The principle is to resect posterior ends of the ribs and bend them in the costal cartilage to form a new roof of the chest. The ribs are fixed posteriorly by nylon or stainless steel sutures through drill holes to the posterior end of the uppermost intact rib. This osteoplastic thoracoplasty is then sized according to the amount and quality

From Sabbatsberg Hospital, Stockholm, Sweden, Surgeon-in-Chief: C. Crafoord, M.D., Professor.

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of the remaining lung tissue at the end of the pulmonary resection for tuberculosis. Good experience with the osteoplastic roof thoracoplasty, now performed in more than 200 cases since 1953, has made me try to utilize the same principle after pneumonectomies.

EARLIER METHODS

1. *Pneumothorax.*—After a pneumonectomy for tuberculosis, the remaining space may be left to become obliterated without thoracoplasty. Especially on the left side where the elevated diaphragm can significantly reduce the space, the herniation of the other lung, with increasing emphysema, may not be so pronounced. Air refills in the remaining space will keep the mediastinum in the mid-line for some time and prevent a too rapid overexpansion of the remaining lung.

To follow this line of treatment the following two criteria must be fulfilled: (a) possibilities for infection in the remaining space must be excluded; and (b) no demonstrable tuberculosis should be found in the remaining lung.

In our material, both these criteria will very seldom be fulfilled at the same time.

2. *Rib resection.*—The most common method of diminishing the thoracic cavity after a pneumonectomy is by rib resection. This will then mean a two-stage procedure. Usually the lung is removed first and then the ribs later on. The simultaneous rib resection has been performed but is not recommended, especially not after an extrapleural resection, where only ribbons of periosteum and intercostal muscles remain of the chest wall after a rib resection. The paradoxical movements of the mediastinum will make it difficult for the patient to raise his sputum, although the procedure is tolerated by good risk patients.

Earlier, we often performed first a five-rib thoracoplasty, which then at a second stage was extended, when the lung was removed. The rib regeneration after the thoracoplasty, however, often made it difficult to obliterate effectively the remaining space after the pneumonectomy in the second stage. Most of these cases therefore have a considerable herniation of the remaining lung into the side upon which the operation had been performed.

3. *Plombage.*—Plastic material to fill the space is now rarely used. This method has caused too much trouble with infection and cavity perforations, when used over lung tissue. When used to fill a space after total pneumonectomy, either intrapleurally (7 cases) or extraperiosteally (5 cases), we have so far not encountered any complications. Only patients with a healthy pleura were chosen in whom there was thought to be no danger of infection.

In my series of 155 cases of pneumonectomies for tuberculosis, I have thus only used plombage to obliterate the space in 12 cases.

THE AUTHOR'S METHOD: AN OSTEOPLASTIC WALL THORACOPLASTY

Indications.—There is always great trauma for the patient, to wake up after a major intrathoracic operation only to look forward to another operation in the near future. Besides the psychologic trauma, the stay in the hospital is prolonged. Furthermore, it is more difficult to regain full arm movements and obtain a perfect healing of the muscles after a second operation.

The osteoplastic wall thoracoplasty after a pneumonectomy gives the patient all the advantages of a one-stage procedure. The immediate obliteration of the remaining space will diminish the incidence of infection. Therefore, in cases of pneumonectomy for tuberculosis, especially in cases of extrapleural resection due to empyema, the operation is completed with an osteoplastic wall thoracoplasty. In 1 case where a tuberculous empyema was in the state of breaking through the chest wall, the space got infected after resection

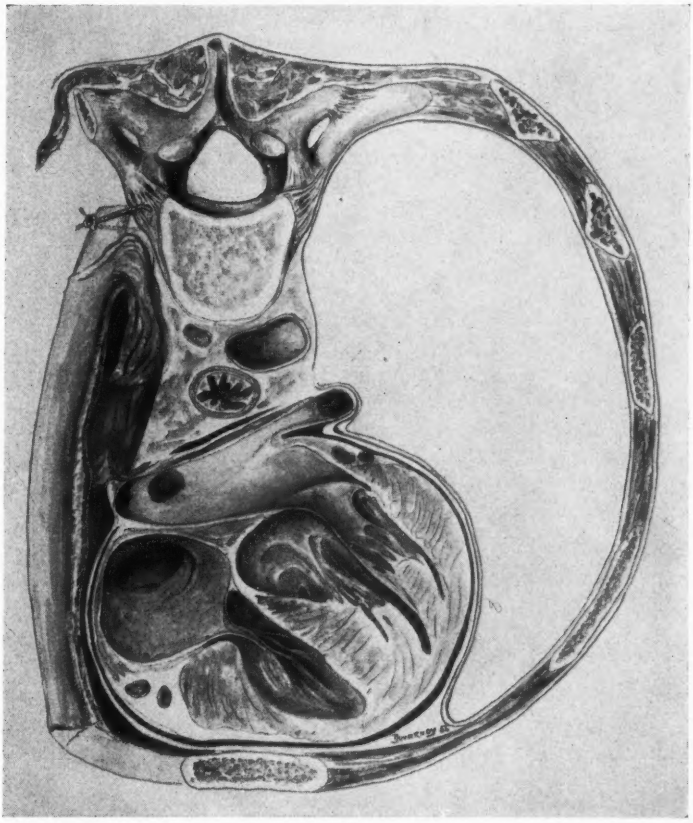


Fig. 1.—The ribs are resected posteriorly. They are then bent or fractured in the costochondral junction, so they lie against the mediastinum. The posterior ends are fixed with a nylon suture through a drill hole in the rib and through the costovertebral ligament.

with osteoplastic thoracoplasty. All of the chest wall except the skin had to be resected. In such a case, it is better to resect the whole chest wall together with the lung at once, utilizing prolonged artificial ventilation for the postoperative treatment.

Technique.—In cases with an accumulation of secretion behind a bronchial stenosis or with empyema and bronchopleural fistula, it is essential to prevent spill over. A left-sided bronchial stenosis will prevent the use of endobronchial

anesthesia with a Carlens tube and then, too, the face-down position, recommended by Overholt, is the position of choice. When one is familiar with the prone position, it will be preferred for all pneumonectomy cases.

In a case with a thick-walled empyema, it is sometimes preferable to do the extrapleural "strip" from the chest wall with the ribs in place. Often some hard work may, however, be saved by first resecting the posterior ends of the upper 9 ribs on the left side and the upper 10 ribs on the right side from the tip of the transverse process. After completion of the pneumonectomy, the osteoplastic wall thoracoplasty is carried out in the following way.

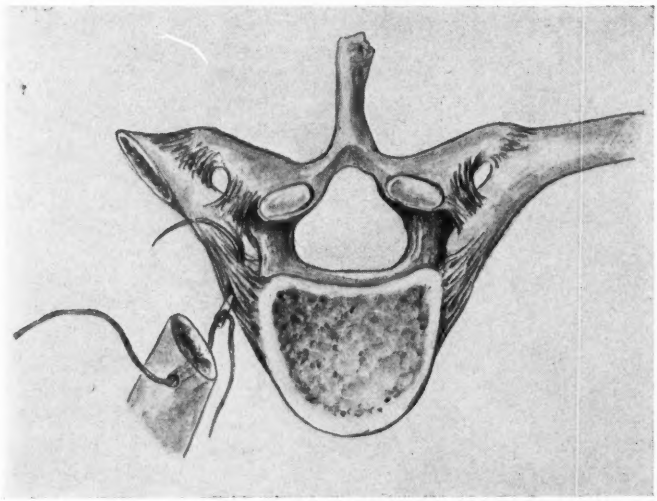


Fig. 2.—The posterior ends of the ribs are fixed to the vertebral body by a nylon suture through the costovertebral ligament and then through a drill hole in the rib.

Rib resection:

a. The entire length of the first rib, including its costal cartilage, is resected to prevent a remaining apical air space. The first rib has only been left in place in absolutely clean cases.

b. Ribs 2 to 9 on the left side and 2 to 10 on the right side are shortened enough to become straight, so that when they are bent or preferably fractured in the costochondral junction, they will reach the vertebral body (Fig. 1).

c. A drill hole (an especially devised drill bore manufactured by Stille, Sweden, is used) is then made in the posterior end of the ribs. The ribs are then fixed to the vertebral bodies by heavy nylon sutures through the drill holes in the ribs, usually through the costovertebral ligament (Fig. 2).

In the beginning, the posterior ends of the ribs were sutured to the anterior longitudinal ligament of the spine but a much stronger fixation is usually obtained by placing the nylon sutures through the costovertebral ligament and behind the tip of the head of a rib (Figs. 1 and 2).

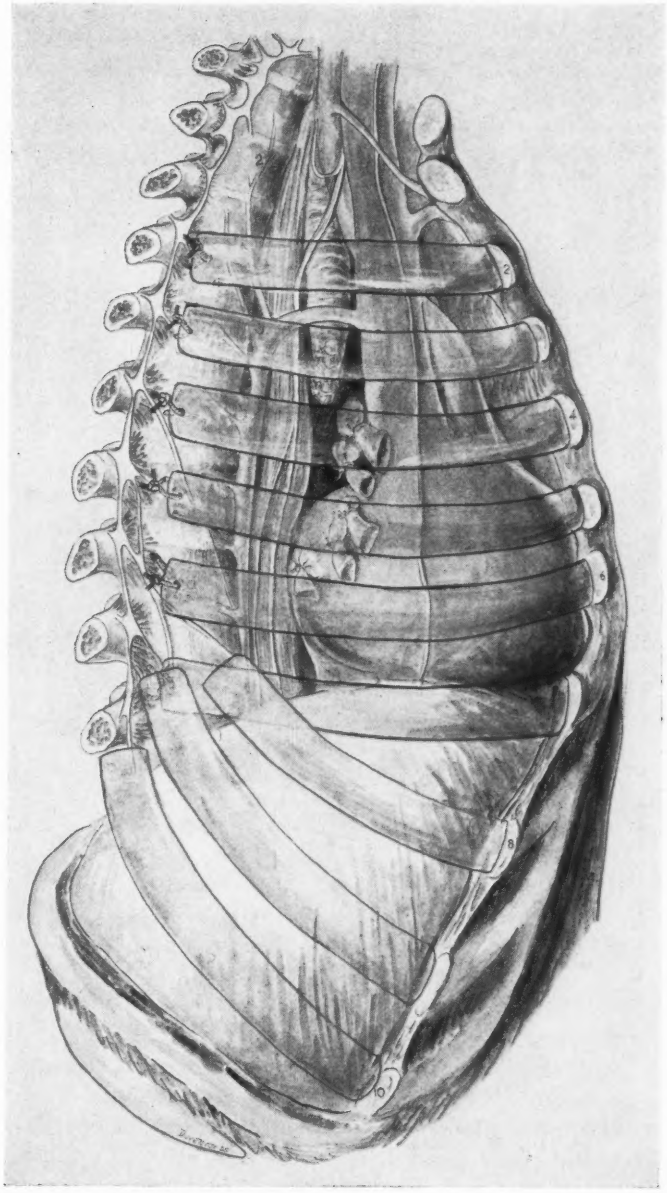


Fig. 3.—The first rib is resected. Then ribs 2 to 7 are bent down and fixed to the thoracic vertebrae 4 to 9. Ribs 8 to 9 on the left side and 8 to 10 on the right side are resected posteriorly but left outside the fixed ribs to round off the contour.

Rib 2 is fixed to the fourth thoracic vertebra.
Rib 3 is fixed to the fifth thoracic vertebra.
Rib 4 is fixed to the sixth thoracic vertebra.
Rib 5 is fixed to the seventh thoracic vertebra.
Rib 6 is fixed to the eighth thoracic vertebra.
Rib 7 is fixed to the ninth thoracic vertebra (Fig. 3).

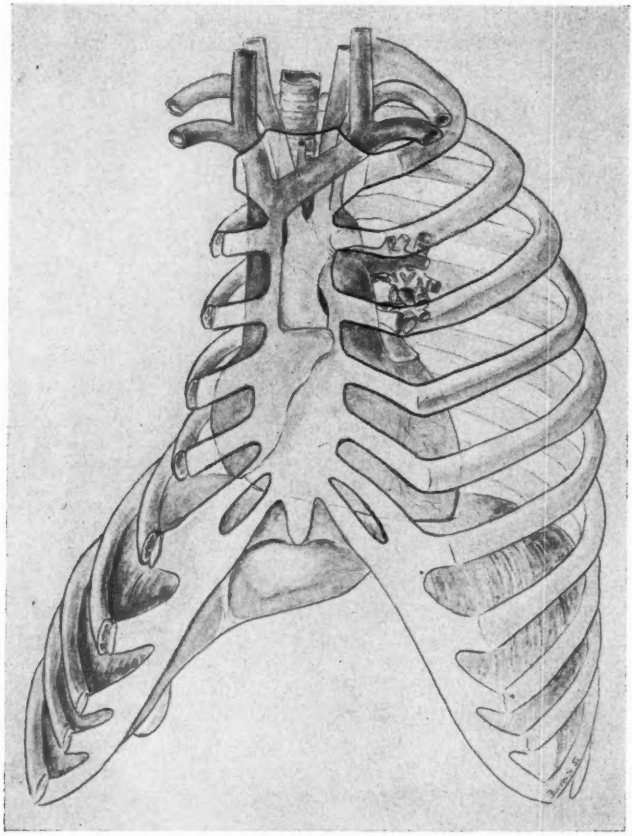


Fig. 4.—A stable wall for the mediastinum is obtained. The space will be obliterated and a herniation of the remaining lung prevented.

When rib 7 or sometimes 8 has been fixed to the lowest possible thoracic vertebra, a stable wall for the mediastinum is obtained (Figs. 1 and 4). All nylon sutures are first placed before they are tied. All intercostal nerves below the fifth intercostal space are left intact as the nylon sutures may pass between the intercostal muscle bundles. The two or three lowermost resected ribs do not have to be fixed but are left outside the fixed ribs to round off the contour (Fig. 4). One tube for drainage is left inside the ribs for two to three days (Figs. 5 through 9, Cases 1 to 3).

With this technique the operation is usually prolonged only by about one-half hour. The operation is well tolerated also in cases with considerable ventilatory insufficiency, provided artificial ventilation is maintained during the first postoperative period and until the patients have regained strength enough for coughing and spontaneous ventilation (Figs. 7 and 8, Case 2). Twenty patients altogether have had a pneumonectomy and simultaneous osteoplastic wall thoracoplasty performed. Three patients are dead: 1 after three weeks due to infection with bronchial fistula, 1 after ten days in bronchopneumonia on the remaining lung (autopsy showed a hematoma on the operated side but no source of bleeding). The third patient died six months after the operation in cor pulmonale with pulmonary edema.

When a pneumonectomy is performed under an old thoracoplasty, it may be difficult to obliterate the space by extended rib resection. The same principle may then be followed. First, the posterior part of the chest wall with rib regenerations is excised. The regenerated ribs should then not be removed one by one but the whole part of the chest wall excised in one block. The anterior parts of the ribs are fractured in the costochondral junction and fixed to the vertebral body in the way just described.

In cases without danger of infection in the remaining space, I have tried to resect the posterior part of rib 2, bent it down and fixed it to the transverse process of the sixth vertebra in order to keep the trachea and the mediastinum in the mid-line. Then rib 4 was resected posteriorly and bent down around the heart and fixed to the transverse process of the ninth vertebra. One year after this operation on a 23-year-old man, a considerable herniation of the remaining right lung had occurred (Case 4, Fig. 10), although the trachea was only slightly displaced to the left. The functional result was very good, maximal breathing capacity was 92 L. per minute and the residual air was only 22 per cent of the total volume. The cosmetic result was excellent. As it is essential in most cases to obliterate the space to prevent infection and to prevent herniation of the remaining lung, this modification is now rarely used. The deformity caused by the osteoplastic wall thoracoplasty has to be accepted if the space is to be obliterated and herniation completely prevented. See Fig. 11, *A*, *B*, and *C* which show a 30-year-old woman, one year after an extrapleural pneumonectomy with osteoplastic wall thoracoplasty on the right side.

CASE REPORTS

CASE 1.—A 46-year-old woman was found to have tuberculous infiltration in the right upper lobe in 1952 and complete atelectasis of the right lung in 1953. The right lung became destroyed behind a stenosis of the right main bronchus (Fig. 5). At operation a right extrapleural pneumonectomy was performed and simultaneously an osteoplastic wall thoracoplasty (Fig. 6).

CASE 2.—A 40-year-old woman had had tuberculous infiltrations in both upper lobes since 1926. A pleurisy on the right side in 1949, resulted in an empyema that persisted for six years. There was bilateral cavitation (Fig. 7). Before operation, the arterial oxygen tension at rest was 55 mm. Hg. The maximal breathing capacity was 23 L. per minute. Seventeen per cent of the oxygen uptake and 23 per cent of the ventilation were on the right side. The patient withstood a right extrapleural pneumonectomy with osteoplastic wall thoracoplasty very well with the aid of prolonged artificial ventilation, and is in a good condition ten months later (Fig. 8).

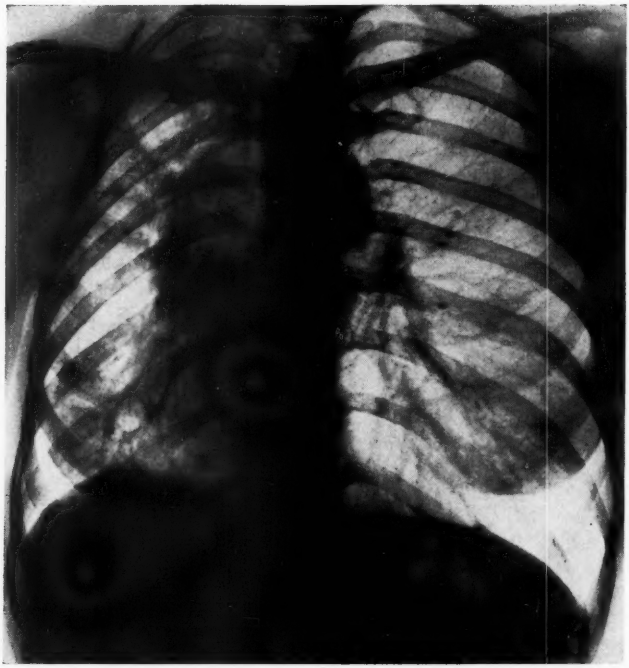


Fig. 5.—Case 1. An x-ray of a 46-year-old woman with stenosis of the right main bronchus and a destroyed right lung.

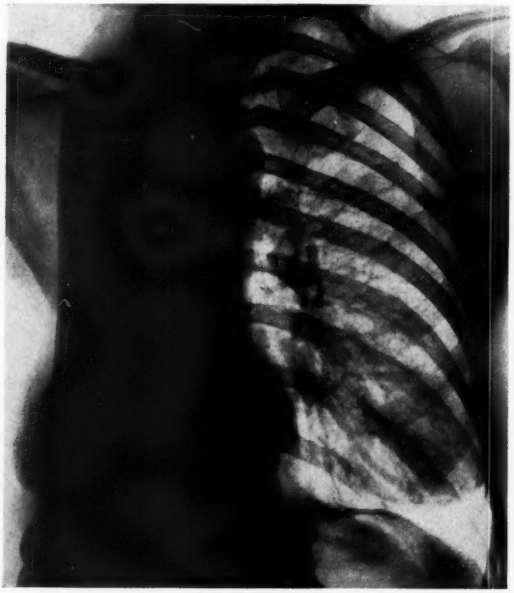


Fig. 6.—Case 1. An x-ray taken three months after an extrapleural pneumonectomy and osteoplastic wall thoracoplasty on the right side.

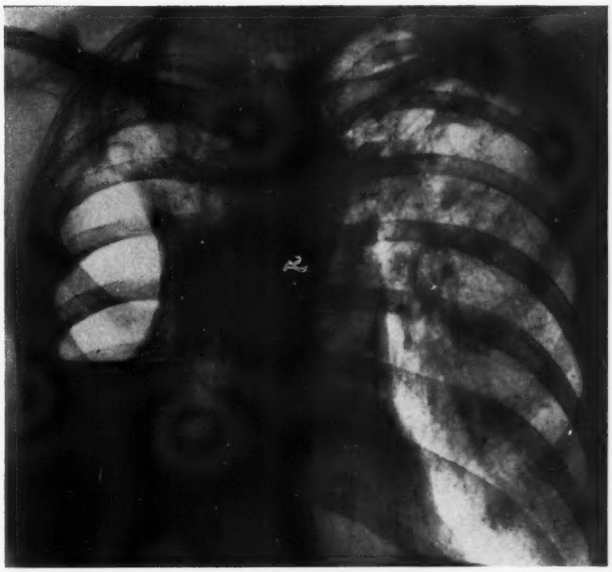


Fig. 7.—Case 2. An x-ray of a 40-year-old woman with bilateral cavitary tuberculosis and a right-sided empyema of six years' duration. Maximal breathing capacity, 23 L. per minute. Seventeen per cent of the total oxygen uptake and 23 per cent of the ventilation came on the right side.

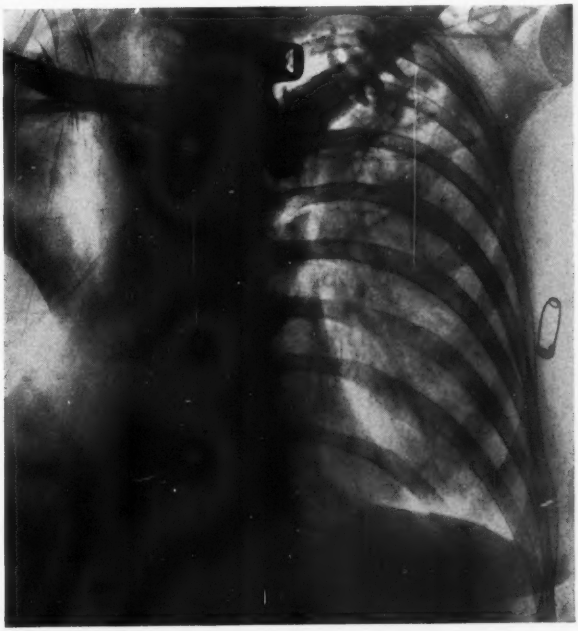


Fig. 8.—Case 2. X-ray taken the day after a right extrapleural pneumonectomy and an osteoplastic wall thoracoplasty. Artificial ventilation was maintained for two weeks. Two days after operation, the patient took over spontaneous ventilation for one hour and then for gradually increasing lengths of time. After three weeks, she could walk slowly around in the ward, and is in good condition ten months later.

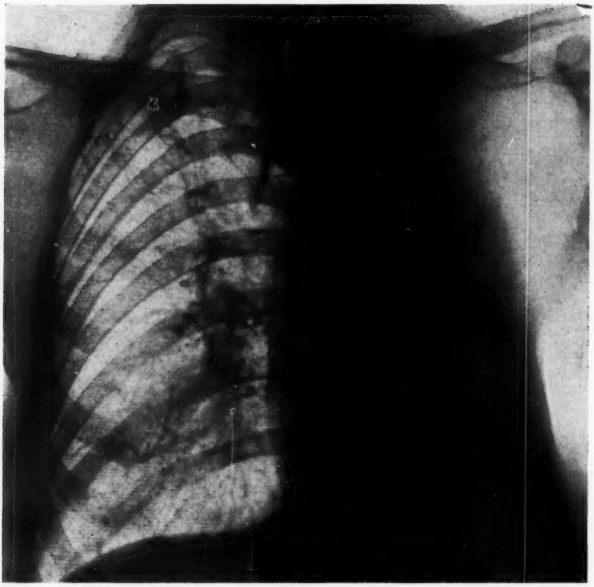


Fig. 9.—Case 3. An x-ray of a 32-year-old man after a left extrapleural pneumonectomy for a destroyed left lung (no oxygen uptake or ventilation on the left side by bronchospirometry) and a tuberculous empyema in an extrapleural pneumothorax space performed eight years earlier.

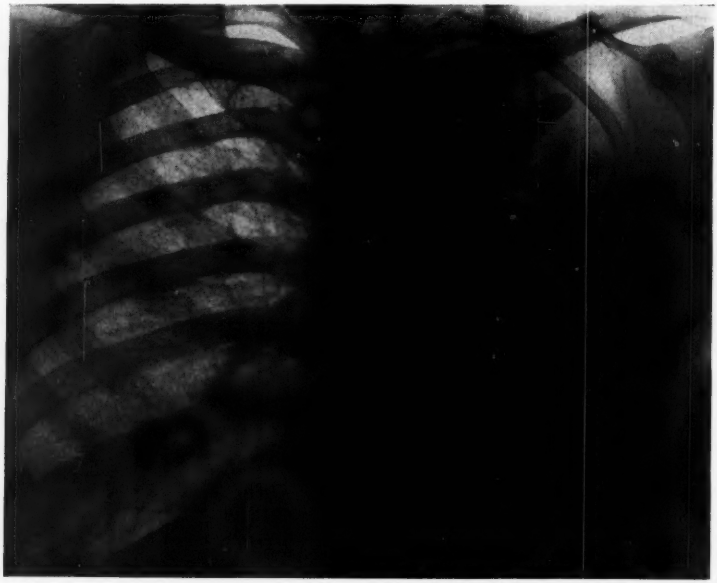
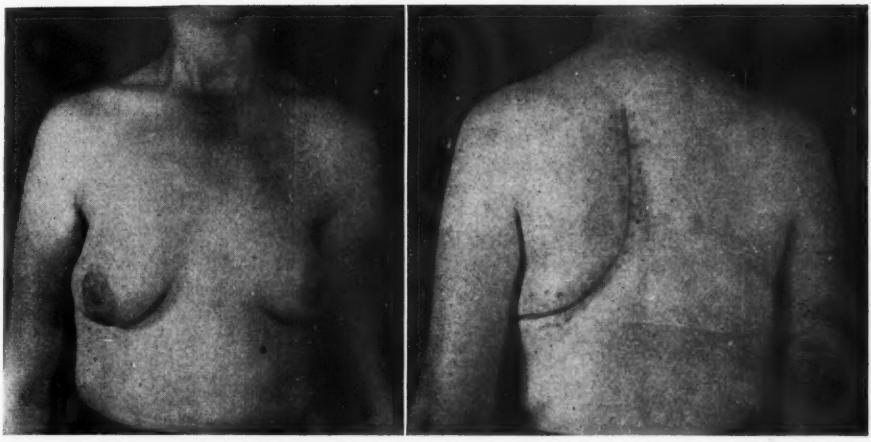


Fig. 10.—Case 4. An x-ray of a 23-year-old man one year after a left pneumonectomy for a destroyed left lung behind a stenosis of the main bronchus. Ribs 2 and 4 were bent down and fixed across the mediastinum. The procedure has resulted in considerable herniation.



A. *B.*

C.

Fig. 11.—*A*, *B*, and *C*. Case 5. The cosmetic result in a 30-year-old woman one year after an extrapleural pneumonectomy with osteoplastic wall thoracoplasty. The first rib was left in place in this case. The patient is working full time. The maximum breathing capacity is 58 L. per minute, the residual capacity 24 per cent of the total. There is only a slight scoliosis. The mediastinum is kept in the midline.

CASE 3.—A 32-year-old man developed a tuberculous cavity in the left upper lobe in 1946. An extrapleural pneumothorax was performed in 1947 but was complicated with a tuberculous empyema. There were, furthermore, remaining cavities in both upper and lower lobes on the left side and in the upper lobe on the right side. The maximal breathing capacity was 50 L. per minute with 100 per cent of the oxygen uptake and ventilation on the right side. An extrapleural pneumonectomy with osteoplastic wall thoracoplasty was performed on the left side (Fig. 9).

CASE 4.—A 23-year-old man had a left pleurisy in 1945. Since 1951, a left main bronchial stenosis had developed with gradual destruction of the left lung. The resting arterial oxygen tension was 87. A left pneumonectomy was performed and at the same time ribs 2 and 4 were bent down across the mediastinum and the heart and fixed to the transverse processes of the sixth and ninth vertebrae. One year after operation, some herniation had developed (Fig. 10). The residual capacity, however, remained 22 L. per minute which was the same as before the operation.

DISCUSSION

A one-stage procedure has been devised to obliterate the space after pneumonectomies. By the immediate obliteration of this space, the danger of infection is diminished which is essential especially in cases with extrapleural resections for empyema.

A stable chest wall against the mediastinum is obtained. This new chest wall will prevent paradoxical movements in the immediate postoperative period. By earlier techniques for total thoracoplasty, it has only been possible to diminish the hemithorax by two-thirds of its original size. Thus, some overexpansion of the remaining lung and a displacement of the mediastinum to the operated side will nearly always follow in spite of the extensive rib resection.

By the new osteoplastic thoracoplasty, the ribs are placed across the mediastinum and fixed to the vertebral body. Thereby, a central position of the mediastinum may be maintained and herniation with overdistention of the remaining lung prevented.

SUMMARY

An osteoplastic wall thoracoplasty has been devised for obliteration of the space after pneumonectomies for tuberculosis in a one-stage procedure.

Paradoxical movements are prevented and the mediastinum maintained in a central position. The procedure has been performed on 20 patients with three deaths.

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HYPOPLASIA OF THE LUNG IN CONGENITAL DIAPHRAGMATIC HERNIA

J. Rakower, M.D., H. Milwidsky, M.D., and P. Wayl, M.D.
Jerusalem, Israel

HERNIAS of the lumbocostal triangle of Bochdalek are the most common type of congenital diaphragmatic hernia encountered in the newborn but are extremely rare in adults.[1, 2] This is due to the fact that most of the infants affected by this condition die in the first days after birth owing to cardiorespiratory failure. Among 50 newborn infants with this type of hernia, 12 survived less than one hour, 9 less than twenty-four hours, and 2 less than thirty-six hours.[3] A review of 210 cases of congenital diaphragmatic hernia revealed that, without operative treatment, 75 per cent of the infants died before the end of the first month of life.[4] Prompt surgical intervention in the postnatal period when the viscera are not yet distended by air is therefore strongly advocated.

However, in some cases the presence of abdominal viscera in the thorax is well tolerated, apparently, and the infant may grow to childhood and even to adult life. The diaphragmatic hernia, in such cases, may be discovered only on routine x-ray examination, as was the case in the patient whose history follows.

CASE REPORT

P. N., a twelve-year-old boy, was admitted to the Department of Chest Diseases of the Hadassah-University Hospital, Jerusalem, Israel, on June 30, 1952. His previous history was uneventful. In October, 1951, on mass radiography, the lower two-thirds of the left thorax was found to be opaque (Fig. 1). This finding was interpreted as pleural effusion and when a pleural tap was attempted, the boy fainted and was admitted to a hospital where he stayed for one week. In June, 1952, pain developed in the left chest accompanied by fever and the boy was admitted to our hospital. On routine examination, the patient appeared normally developed for his age but was poorly nourished. His weight was 28 kilograms and his height 133 cm. Mild scoliosis was noted. The temperature was 36.9° C. to 37.5° C., the pulse 80, the blood pressure 100/65, respirations were 20 per minute, and the erythrocyte sedimentation rate was 60/75. Two weeks later the temperature and erythrocyte sedimentation rate became normal. Hyperresonance to percussion over the left chest was found with absence of normal vesicular breathing and vocal fremitus. The heart sounds were normal. The liver and spleen were not palpable. A blood count showed 4.13 million red blood cells, 12 Gm. hemoglobin, 10,200 white blood cells with 55 per cent neutrophils, 39 per cent lymphocytes, 5 per cent eosinophils, and 1 per cent monocytes. Urine analysis was normal. Serologic tests for syphilis were negative. The Mantoux test in 1/1,000 was positive. Gastric content smears and cultures were negative for acid-fast bacilli. An electrocardiogram revealed right axis deviation. The pulmonary function studies showed the vital capacity to be 40 per cent of the predicted value and the maximal breathing

From the Departments of Chest Diseases and Thoracic Surgery, Hadassah-University Hospital, Jerusalem, Israel.
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TABLE I. VENTILATORY FUNCTION TESTS

	AGE	HEIGHT (CM.)	WEIGHT (KG.)	VITAL CAPACITY OBSERVED (ML.)	PER CENT OF PREDICTED VITAL CAPACITY	TIDAL AIR (ML.)	INSPIRATORY RESERVE (ML.)	EXPIRATORY RESERVE (ML.)	MAXIMUM BREATHING CAPACITY OBSERVED (L./MIN.)	PER CENT OF PREDICTED MAXIMUM BREATHING CAPACITY	AIR FLOW VELOCITY INDEX
Before operation	12	144	30	1030	40	200	531	350	43	47	1.17
After operation (2 months)	12.5	146	31	775	31	200	400	175	30	31	1
After operation (2½ years)	15	166	46	2080	48	673	717	690	71.2	60	1.25

capacity 47 per cent of the predicted value (Table I). Bronchoscopy showed a marked narrowing of the openings of left main and particularly of the left upper and lower lobe bronchi. With bronchoscope No. 6, it was impossible to enter farther than the left-main bronchus.

Chest x-ray revealed the presenec of air in the upper part of the opacity in the left thorax and diaphragmatic hernia was therefore suspected (Fig. 1). A barium meal and a barium enema showed that all the small intestine, cecum, and colon were in the left

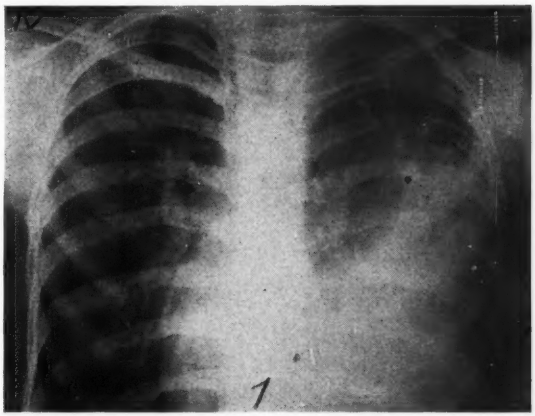

Fig. 1.—Chest x-ray showing an opacity in the lower part of the left thorax due to herniated intestines.

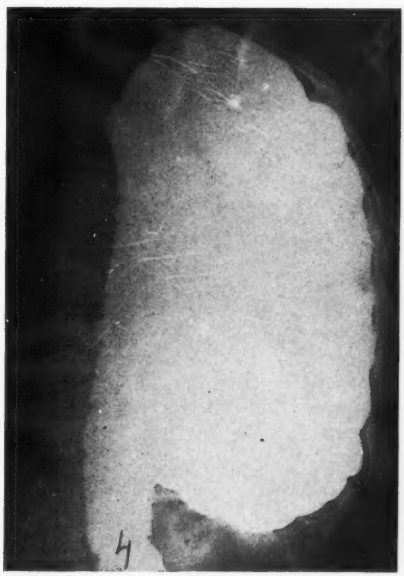

Fig. 2.—X-ray studies after a barium enema to demonstrate the left thorax filled with bowels.

thorax (Figs. 2 and 3), and that the communication between the thorax and abdomen was in the posterior part of the thorax. The left diaphragm was not seen on x-ray and no movements were visible on fluoroscopy. However, a diagnostic pneumoperitoneum (Fig. 4) revealed diaphragmatic movements on screening. Bronchography showed very poor filling of the left bronchial tree, characteristic of hypoplastic lung (Fig. 5). On pyelography, no pathologic changes were found.

Fig. 3.—Roentgenogram after barium meal showing the stomach low in the abdomen and small bowel loops in the left chest.

Fig. 4.—Roentgenogram taken after induction of pneumoperitoneum demonstrating pneumothorax on the left side.

The clinical diagnosis was congenital diaphragmatic hernia through the lumbocostal triangle with hypoplasia of the left lung.

Operation.—(H. M.) Under endotracheal anesthesia, the left thorax was opened through the seventh intercostal space. The pleural cavity was almost completely filled with intestinal loops without parietal peritoneal coverings. The left lung was the size of that seen in a 2-year-old child but showed normal development of both lobes. The lower lobe was atelectatic but could be inflated by positive pressure. The defect in the diaphragm was found in its posterolateral part and measured 10 cm. by 6 cm. Through this defect nearly all of the small bowel (from the upper jejunum to the lower ileum) and the ascending, transverse, and descending colons protruded from the abdominal into the chest cavity. There were many adhesions between the intestines and the margins of the diaphragmatic defect, and these had to be severed before the bowel could be replaced into the abdomen. The defect in the diaphragm was repaired by two rows of mattress sutures. The left phrenic nerve was crushed. The hypoplastic lung was inflated by the anesthetist but filled only one-third to one-half of the thoracic cavity which was then closed with underwater-seal drainage.

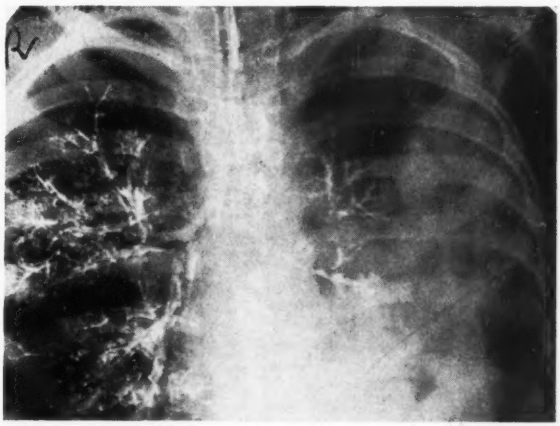

Fig. 5.—Bronchogram demonstrating poor filling of the left bronchial tree.

The postoperative course was somewhat difficult during the first week owing to marked abdominal distention which required the use of Wangensteen suction. Thereafter, however, normal bowel function was resumed and the boy became free of abdominal discomfort.

After operation, the left lung expanded a good deal and vesicular breathing, although weaker than on the right side, became audible, particularly in the upper part of the hemithorax. This expansion was confirmed by a second bronchography (Fig. 6) which showed good filling of the upper and lingular bronchi. However, the lower bronchus still filled poorly. The hypoplastic lung did not fill the entire hemithorax and six weeks after operation a pleural effusion appeared. The pleural dead space, created after the intestines were replaced in the abdomen, was thereafter filled by fibrinous organization of the pleural fluid, elevation of the diaphragm, and mediastinal shifting. Two months after operation, respiratory function tests showed a decrease in vital capacity of 9 per cent and of the maximum breathing capacity of 16 per cent, the result of pleural involvement and phrenicolysis. Two and one-half years after operation, the boy was well and x-ray showed good expansion of the lung (Fig. 7). Pleural thickening was moderate, vital capacity was 48 per cent, and maximum breathing capacity 60 per cent of the predicted values (Table I).

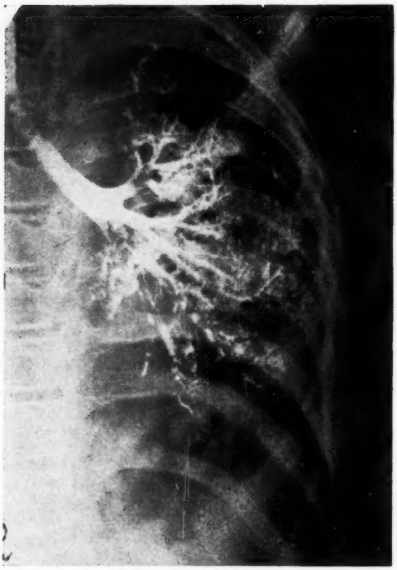

Fig. 6.—Bronchogram taken eight months after operation showing a good filling of the bronchi of the left upper lobe and poor filling of the lower lobe.

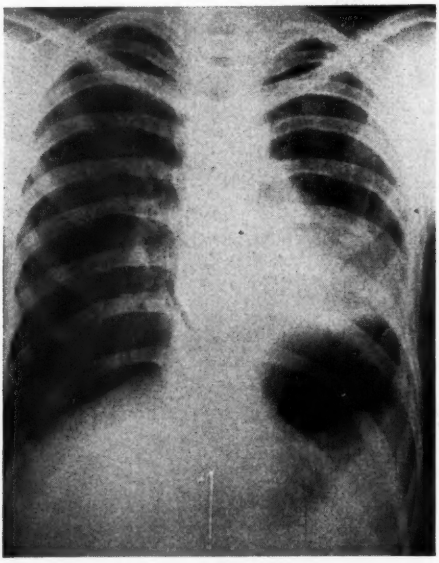

Fig. 7.—Roentgenogram taken two and one-half years after operation showing good re-expansion of the left lung.

DISCUSSION

Although the subject of congenital diaphragmatic hernia is extensively dealt with in surgical literature and the intestinal complications are well known, little attention has been paid to the state of the lung in this condition. In hernia of the foramen of Bochdalek, the lung and mediastinum are usually compressed by the large mass of abdominal viscera which has entered the thorax and only few infants escape early death. If complications from congenital diaphragmatic hernia arise during childhood and adolescent life, they are due mostly to disturbed bowel function such as intestinal obstruction, strangulation, etc., and this may account for the fact that little mention is made of the fate of the lung in this condition. In addition, many surgeons employ and advocate exclusively the abdominal approach for the repair of these diaphragmatic hernias, particularly on the left side. They believe that reduction of the viscera is easier by traction from below than by pulsion from above.[6] It is evident that the use of the abdominal approach precludes proper visualization and palpation of the lung and, thus, pulmonary pathology will escape detection and adequate treatment. Other authors recommend the thoracic approach because of the better exposure obtained, the possibility of more rapid expansion of the lung, and the much smoother convalescence.[7-9] We employ the thoracotomy approach on hernia of foramen of Bochdalek and find it superior to the abdominal approach. We feel that the lung should be carefully examined in every case and that inflation should be instituted as soon as the eventrated viscera have been returned to the abdominal cavity.

We have found only two instances in the literature where the state of the lung in congenital diaphragmatic hernia is mentioned. Bonilla-Naar and co-workers[5] report on a 10-year-old boy who complained of dyspnea and in whom diaphragmatic hernia was diagnosed. At operation, a completely atelectatic left lung was found. The upper lobe was inflated but the lower lobe remained airless and functionless. These authors used the lower lobe to plug the large defect in the posterolateral part of the diaphragm. Fisher[10] reported on a 12-year-old girl with diaphragmatic hernia in whom the bronchogram showed a filling defect in the left lower lobe. During closure of the diaphragmatic defect, an atelectatic and hypoplastic lower lobe was found.

In our case, the hypoplastic lung was inflated and its function partially restored. The postoperative x-ray studies and ventilatory function tests showed a marked improvement in pulmonary function in spite of phrenic interruption and pleural fibrosis.

SUMMARY

A case of congenital diaphragmatic hernia through the foramen of Bochdalek with hypoplasia of the lung is reported. The problem of the state of the lung in cases of diaphragmatic hernia is discussed and the thoracic approach for the repair of these hernias is recommended.

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PANCOAST TUMOR

Five-Year Survival Without Recurrence or Metastases Following Radical Resection and Postoperative Irradiation

William M. Chardack, M.D., Buffalo, N. Y., and James D. MacCallum, M.D., Warsaw, N. Y.

PAIN, paralysis, and atrophy within the lower brachial plexus territory, Horner's syndrome, and a density in the pulmonary apex with erosion of adjacent ribs are the salient features of the Pancoast syndrome. Although similar isolated cases had been described prior to Pancoast's original publication in 1924,[12] he was the first to draw wide attention to the entity now bearing his name.

Three typical cases formed the basis of his report. A biopsy had been taken in 2 of the 3 cases and had revealed a malignant tumor; death had occurred rapidly in all 3 from what he termed a "comparatively trivial growth without detectable metastases."

Pancoast thought he was dealing with a new and specific clinical entity, a carcinoma arising from epithelial rests of the fifth branchial pouch.

In 1932, addressing the Radiological Section of the American Medical Association, he[13] reported 4 additional cases, suggested that the lesion be called "superior sulcus tumor," and restated his belief in its branchial origin although he prudently added: "It is possible that this designation may be changed again with a better knowledge of the histopathology of the growth."

Herbut and Watson[8] reviewed the literature on this subject in 1946, collected 151 cases, concluded that a variety of malignant tumors can indeed produce this syndrome and proposed the comprehensive designation of "tumor of the thoracic inlet producing the Pancoast syndrome." In 1953, in our preliminary report on this case,[3] we reviewed all contributions to this subject published subsequently to Herbut and Watson's study and this perusal of the world literature has been brought up to date. Mention will be made in the text only of those publications dealing with new and pertinent facts. For a detailed description of the syndrome, the reader is referred to Herbut and Watson's article. We shall limit ourselves to a summary of the present concept of this lesion and a follow-up report on a five-year survival and possible cure following removal of such a tumor.

The anatomic location of this lesion within the confines of the thoracic inlet determines its clinical features; the latter are the result of compression and invasion of the brachial plexus, sympathetic chain, and the surround-

From the Department of Surgery, University of Buffalo School of Medicine and the Surgical Service, Veterans Administration Hospital, Buffalo, N. Y.

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ing ribs. Depending upon size, stage of development, and invasive qualities of the lesion, the syndrome may be incomplete or additional features may appear as further neighboring structures, vascular or other, become engulfed by the tumor.

With regard to the origin of the lesion, Pancoast's concept of a specific branchiogenic epithelioma was discarded as subsequent observations showed that a wide variety of malignant tumors may produce a Pancoast syndrome, either by direct extension into or by metastases located in the thoracic inlet. However, the vast majority of such cases are due to a bronchogenic carcinoma arising in the extreme pulmonary apex.

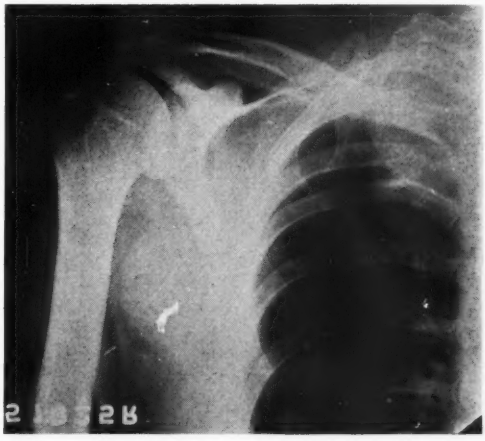
There has been unanimity of opinion in the past on the rapidly fatal course of these tumors. Indeed, it is widely held that the lesion is an incurable one and that the presence of a Pancoast syndrome signifies inoperability and prohibits surgical exploration.

Past experience, it is true, had justified the pessimism expressed with regard to the prognosis of a Pancoast syndrome. Herbut and Watson[8] stated in 1946, "to date the disease has been 100 per cent fatal. Roentgen therapy so far appears of no avail." They suggested, however, that in the future an operable case might be detected and urged thorough investigation, even by surgical exploration if necessary, of any persistent neuritic pain of the upper limb. Walker,[14] in 1946, wrote "death has occurred in all cases recognized up to the present time. In no instance has it been possible to remove the tumor surgically and irradiation has not been of palliative benefit."

A review of the literature up to the date of this publication has failed to yield any authenicated cases of a successful resection of such a tumor with the exception of the 1 reported by the authors in 1953. Actually only seven explorations of these lesions had by then been recorded, and in no instance had the tumor been resected. More recently, Becker,[1] in 1953, recorded an exploratory thoracotomy but the tumor was nonresectable. Hamm,[7] in 1952, stated that only operative treatment might improve the prognosis and he quoted Frey who is said to have reported a successful surgical resection of such a tumor to the Congress of Internal Medicine in 1951. Frey's[5] address, however, does not contain any reference to a Pancoast tumor. The presentation dealt with recent advances in thoracic surgery and made mention only of a case of a sarcoma of the lung in the dependent portion of the left upper lobe with invasion of the chest wall in this lower area.

While the experience with x-ray treatment of this lesion had also been uniformly disappointing in the past and the majority of reports indicated that it had been of no avail, there have been some recent exceptions to this view. Hamm[7] records a case of undifferentiated epithelioma producing a Pancoast tumor and proved by biopsy. He states that delivery of 2,800 r led to a surprise remission which had lasted for more than one year at the time of his publication and the patient was then still under observation. Haas and associates,[6] in 1954, stated that they had relatively better success with Pancoast type lesions than with other bronchogenic neoplasms. They had treated 4 patients with such tumors and, of these, 1 was living and well thirty-

four months after irradiation. There had been regression of the mass as well as recalcification of the rib after delivery of 11,063 r by betatron therapy. No details are given with regard to histologic proof of the nature of the lesion. Miceli,[11] in 1952, stated that he had obtained some temporary relief by x-ray therapy.



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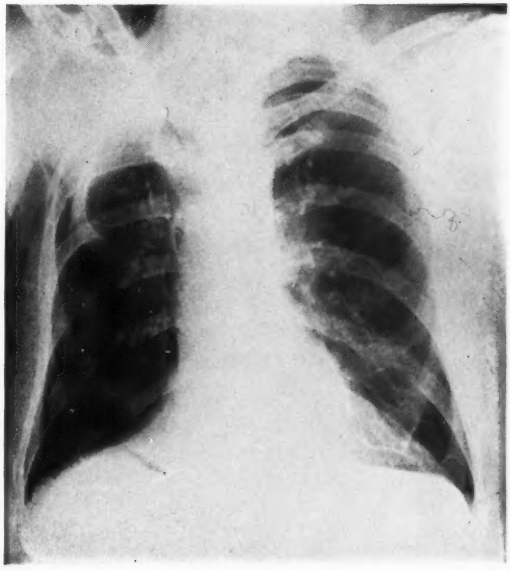


B.

Fig. 1.—X-ray photograph of the right shoulder and apex of the lung at time of admission (*A*) and five years after operation (*B*). (*A*, From Chardack and MacCallum, J. Thoracic Surg., April, 1953.)

Malcolmson,[9] in 1947, reported a case in which irradiation had produced the regression of a large mass in the left upper lobe. The patient had been followed for eighteen years and the tentative diagnosis suggested by the Mayo Clinic was one of lymphoblastoma. Formal proof by biopsy had not been obtained.

It may then be stated that only a very few patients with Pancoast tumors have had exploratory operations, that we know of no other instance of a successful resection, and that in the opinion of the majority of writers, x-ray therapy has been of no avail in proved carcinoma with the exception of the previously quoted instances in none of which, however, has it produced an authenticated cure in a proved malignant tumor.

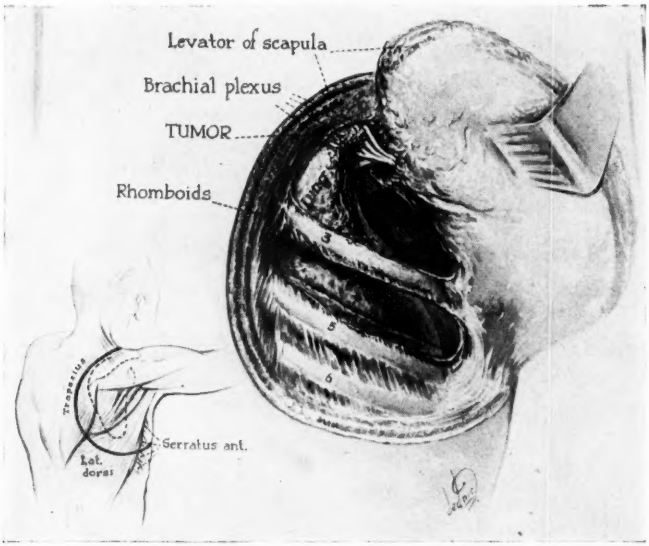
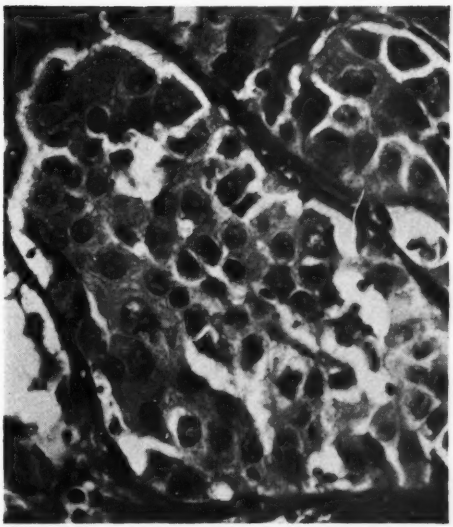


Fig. 2.—Artist's conception of the operative field and the line of incision. (From J. Thoracic Surg., April, 1953.)

CASE REPORT

H. R., a 54-year-old white man, was admitted to the Veterans Administration Hospital in Buffalo, N. Y., on April 19, 1950, complaining of excruciating and intractable pain around the shoulder which radiated into the ulnar territory. A paralysis of ulnar distribution was present. There were ptosis and myosis of the right eye and an x-ray of the chest showed a small density in the right apex (Fig. 1, *A*). Additional laboratory findings were noncontributory. The clinical diagnosis was apical bronchiogenic carcinoma with Pancoast syndrome and surgical intervention was carried out on May 15, 1950.

Operative Procedure.—For a detailed description of technique, the reader is referred to our original report in 1953.[3] Suffice it to say here that through a long periscapular incision, the tumor was removed in toto by a right upper lobectomy and en bloc excision of the involved ribs, pleura, endothoracic fascia, and spinal nerve roots at the level of the first and the eighth thoracic vertebrae, and the seventh cervical vertebra. Fig. 2 shows surgical exposure and the tumor extending through the apex of the pleural dome into the brachial plexus and it was felt that by sacrificing the invaded roots, there was a reasonable chance to remove the



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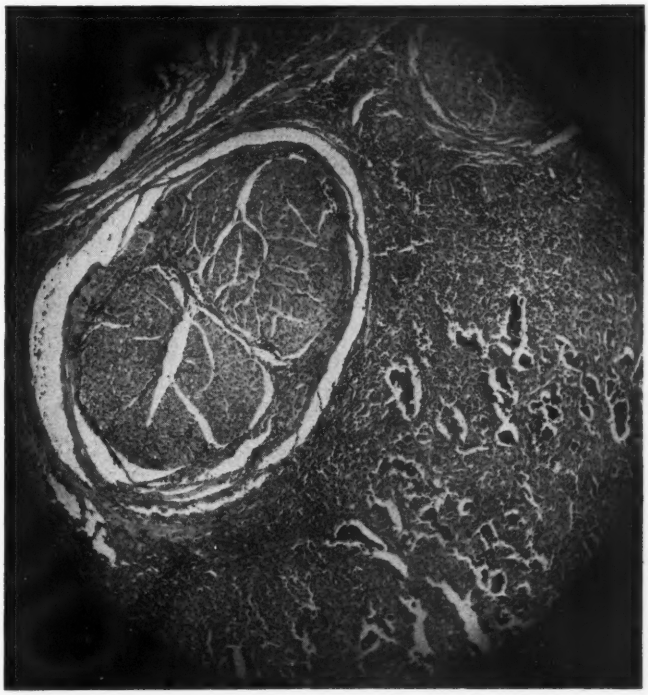
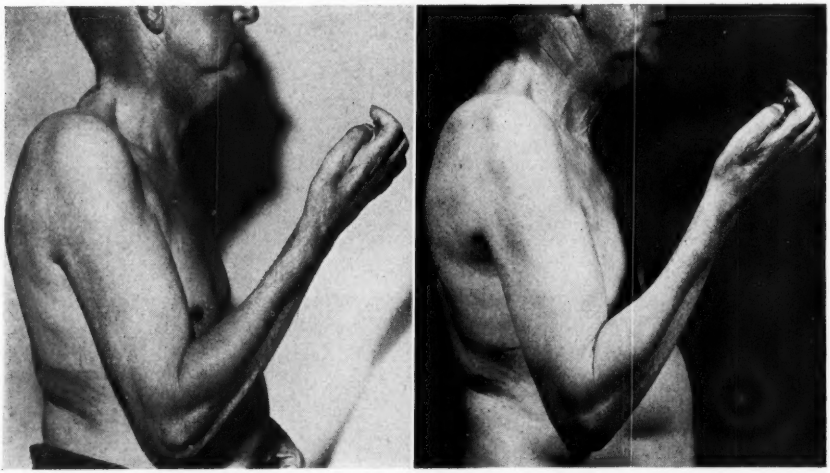


B.

Fig. 3.—High power microphotograph of the tumor tissue from the intrapulmonary component (*A*), and microphotograph showing invasion of a brachial plexus nerve (*B*). (From J. Thoracic Surg., April, 1953.)

entire growth. Pathologic examination of the specimen revealed a highly undifferentiated squamous-cell carcinoma, clearly originating in and traceable to one of the minor branches of the right upper-lobe bronchus. Invasion by the tumor of the perineural spaces of the brachial plexus cord is demonstrated on the photomicrograph in Fig. 3.

Postoperative Course and Follow-up.—The postoperative course was uneventful. Neurologic examination revealed the expected deficit due to removal of roots at the level of the seventh and eighth cervical vertebrae and the first thoracic vertebra but the postoperative changes were hardly more extensive than the damage from tumor involvement observed preoperatively.



A. *B.*

Fig. 4.—Photographs of the patient in 1952 (*A*), and in April, 1955 (*B*). (*A*, From J. Thoracic Surg., April, 1953.)

Although recorded experience with radiotherapy had been disappointing, it was felt that, in this instance, with all gross carcinoma removed and the overlying bony structures resected, the possibility of delivering a cancerocidal dose to any potentially remaining microscopic foci of an undifferentiated tumor was greatly enhanced. Accordingly, three weeks after surgery, radiation therapy was begun and 6,528 r (half value layer copper 25 mm.) were delivered over fifty-four days to the general tumor area.*

The patient was followed at approximately three-month intervals and his present status five years after the operation is as follows: he weighs approximately one hundred seventy pounds, ten pounds more than before the operation and his weight has been stable since 1951. His general condition is excellent. There is no evidence of recurrence or metastasis and there has been no increase in the extent of his paralysis. Trophic changes of the tissues of the hand are marked. He complains of an occasional mild, burning sensation in the right hand but grades this as negligible compared to the pain sensation before the operation. This slight discomfort is entirely acceptable to the patient and he does not require medication. There is no interference with sleep. He has adjusted to the disability resulting from the paralysis and feels that the limb is still partially useful to him. He was last examined in April, 1955.

*Therapy was carried out by Dr. Walter T. Murphy, Roswell Park Memorial Institute, Buffalo, N. Y., Consultant Radiotherapist to the VA Hospital, Buffalo, N. Y.

DISCUSSION

Since, in the view of many surgeons, it is widely believed that the presence of a Pancoast tumor signifies inoperability and incurability and constitutes a contraindication to exploration, we feel that, in the light of our experience, this concept deserves revision. It has been shown that a Pancoast tumor, or more specifically, that a bronchogenic carcinoma with Pancoast syndrome has been successfully treated by radical resection of all involved structures, followed by radiotherapy. Several aspects deserve further discussion.

Of particular interest is the question as to whether a Pancoast syndrome is invariably and exclusively due to a malignant tumor or whether a benign tumor or even a nonneoplastic lesion may on occasion be responsible for a similar symptom complex. Both questions can be categorically answered in the positive since our review of the literature, in 1953, yielded 6 such authenticated cases. In 1 instance, a neuroma was responsible for the syndrome, tuberculosis accounted for 3 and a Hand-Schüller-Christian disease and a nonspecific pneumonitis of the left upper lobe for the remaining 2 cases. Since then, similar experiences have come to light. D'Eschougues and Houel,[4] in 1954, reported a Pancoast syndrome due to the presence of a fist-sized hydatid cyst removed by thoracotomy. Bellion,[2] in 1953, and Marques and associates,[10] in 1952, reported the presence of a Pancoast syndrome due to Hodgkin's disease proved by biopsy and treated radiologically. The importance of these observations is considerable since they indicate that it is the location and not the nature of the expanding lesion that is responsible for the genesis of the syndrome. Far from contraindicating surgical exploration, the presence of a Pancoast syndrome imperatively dictates intervention since an entirely benign lesion and one not necessarily amenable to radiotherapy may be its cause.

The question may be raised as to the part played by the radiation therapy in the cure of our case. In past experience, radiotherapy alone has not been capable of achieving a cure of a Pancoast syndrome due to a proved malignant tumor. Radical surgical removal followed by intensive radiotherapy may well be more potent than either method alone, when one is dealing with a tumor showing invasion of preformed and lymphatic spaces such as was the case here. It seems logical to assume that while all grossly recognizable tumor tissue with an apparently adequate margin of normal tissue is removed by radical surgery, microscopic foci may remain beyond the field of excision and it is conceivable that a cancerocidal dose delivered to a relatively wide field may be of value, especially when overlying soft tissues and bony structures have been removed. In any event, the combination of extensive removal followed by radiotherapy was capable of dealing with a lesion heretofore considered to be always rapidly fatal.

In addition to the now proved possibility of achieving at least a five-year survival without demonstrable recurrence or metastases, surgical removal is capable of producing important palliation in a disease notorious for the unbearable pain and discomfort it produces. Even if not entirely removable, partial ablation of a tumor in this particular location is justifiable since it can produce impressive pain relief. Since our original communication in 1953,[3] we

have had occasion to perform an incomplete removal and decompression of a Pancoast tumor in 3 additional cases. One of these patients died a few months after operation but 2 of the patients are still under observation, although only a few months have elapsed since surgical intervention. In both, radiotherapy was directed to the remaining small and unresectable portions of the tumor, and palliation has been remarkable.

SUMMARY AND CONCLUSIONS

1. A follow-up report on a case of Pancoast syndrome due to bronchogenic carcinoma is presented. This lesion, generally thought to be inoperable, has been successfully treated by en bloc resection of the tumor and adjacent involved structures, followed by irradiation. The patient is now well and free of recurrence and metastases five years after the procedure.

2. The prevailing concept that the Pancoast syndrome signifies inoperability and incurability should be reconsidered and any unexplained persistent neuritic pain in the upper limb should be thoroughly investigated with the possibility of a Pancoast syndrome-producing lesion in mind. Earliest surgical exploration is justified because: (a) the lesion may be benign and not necessarily radiosensitive, (b) if it is malignant, it may be resectable and possibly be curable, and (c) if it is not resectable, important palliative benefits may be achieved by decompression in an otherwise hopeless and extremely distressing type of tumor.

Addendum: The patient was seen last at this hospital in February, 1955. He was well. On March 13, 1956, he died after a brief illness, five years and ten months after his operation. A post-mortem examination was granted and no evidence of recurrence or metastases was found. The thoracic organs as well as the contents of the right supraclavicular triangle were dissected grossly and studied microscopically by Dr. Kornel L. Terplan, Professor of Pathology, University of Buffalo School of Medicine. The cause of death was bronchopneumonia, mild cor pulmonale, and myocardial failure. There was no evidence of tumor.

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THE PROBLEM OF CARCINOMA IN ACHALASIA

STANLEY E. POTTER, M.D., JOHN A. RASMUSSEN, M.D., AND
R. RUSSELL BEST, M.D.
OMAHA, NEB.

THOSE who explore for the first time the literature on this interesting subject are met immediately by a number of paradoxes, most of which are difficult to resolve.

The reported number of cases is remarkably small, but more remarkable is the fact that certain cases have been excluded from a review of the literature, "where it has not been possible, owing to wartime dispersal of medical libraries, to study the original article."[1] Other cases have not been included in tabulated reviews because of "insufficient clinical details."[1]

We feel that to perpetuate the figure of 15 or 16[2, 3] reported cases accomplishes nothing, for reported cases are no index of occurrence particularly when their very numbers are suspect.

Many more imponderables arise. Gottstein[5] reported the incidence of carcinoma in achalasia in his series at 10 per cent; Rake[6] reported an incidence of 20 per cent. Poles apart, statistically, is Vinson[7] who makes no mention of carcinoma in reporting 683 cases of achalasia.

When one approaches the problem from the aspect of esophageal carcinoma, essential agreement with infrequency is found. In an experience with over 600 cases of carcinoma of the esophagus, Puestow had none associated with achalasia.[8]

Attempts to resolve these opposites have the appeal of rationalizing that the European incidence is higher, or that our identification of this disease association is careless or not recorded. We have no proof of the former but we believe the latter possible, in our own geographic area at least.

Much stress has been placed upon repeated esophagoscopic examinations in achalasia to detect the development of carcinomatous change. This is indeed worthy. We feel also that the periodic esophagogram done by an experienced radiologist in a well-prepared esophagus will be useful and may provide the necessary periodic follow-up for more patients. In reviewing the assembled cases, we were impressed by the high incidence of positive radiologic diagnoses of tumor, if the possibility were kept in mind.

Stimulated by a recent case of our own, we have reviewed all detailed cases in the English literature in an effort to find an early clue to this formidable complication of achalasia (Table I).

From the University of Nebraska College of Medicine, Omaha, Neb.
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TABLE I

AUTHOR	SEX	AGE	DURATION OF ACHALASIA	ASPIRATION PNEUMONIA	BLEEDING	ANEMIA	WEIGHT LOSS	CHANGE IN HISTORY
Sicher[9]	F	47	10 years	None	Not stated	Yes	"Several pounds"	Increase in severity and loss of intermittency of dysphagia
Kornblum and Fisher[10] Case III	F	47	10 years	Questionable	Yes	Yes	Yes	Not stated
Kornblum and Fisher[10] Case IV	M	46	18 years	None	Not stated	Not stated	Yes	Cough and weight loss
Vinson and Kennedy[7]	M	34	8 years	None	Not stated	Not stated	Yes	Progressive dysphagia
Bersak[4]	M	47	10 years	None	Not stated	No	Yes	Progressive dysphagia
Baer and Sicher[1]	M	65	20 years	None	Yes; vomitus	Not stated	Emaciation	Increasing dysphagia; hematemesis
Gore and Lam[2]	M	42	10 years	None	Yes; vomitus	Not stated	30 pounds	Hematemesis; return of dysphagia on conservative management
Kastl[3]	M	56	24 years	Yes	Not stated	Yes	30 pounds	Chills, fever, productive cough
Potter, Rasmussen and Best	M	51	4-5 years	Yes	No	No	20 pounds	Progressive dysphagia

The sex incidence, as with carcinoma of the esophagus alone, is predominantly male. The age incidence is that of carcinoma in general.

It would appear that carcinoma favors long-standing achalasia; the average duration of proved or suspected esophageal dilation being twelve years. This fact, along with the low incidence of tumor, is difficult to reconcile with the belief that chronic irritation is important in the development of epidermoid carcinoma.

Aspiration pneumonia, probably a later manifestation and what first brought our patient to seek medical aid, was not common in the reviewed cases.

Bleeding, either hematemesis or melena, is not usual in uncomplicated achalasia. Though associated with other complications, its appearance with achalasia demands a careful search for carcinoma. The presence of secondary anemia requires the same attention.

Weight loss has been consistent in all cases and must not be explained carelessly as being due to the existing disease.

The major pitfall in the diagnosis is dysphagia, this sign being cardinal in both carcinoma and achalasia. In the former, we have believed it to be due to the mechanical influence of the progressive narrowing of the lumen of the esophagus. We were surprised to find that annular carcinomatous involvement in achalasia was rare, recognizing that the distance "traveled"

TABLE I

X-RAY EVIDENCE OF CARCINOMA	ESOPHAGOSCOPY	LOCATION OF TUMOR	SURGERY	SURVIVAL	COMMENT
Constriction, middle third	Ulcerating growth; not annular	Junction middle and distal thirds	Esophagogastrectomy	2 days	Epidermoid tumor
No	Fungating mass; not annular	Proximal part of distal third	Not stated	Not stated	Squamous-cell, grade III
Yes	Fungating mass; not annular	Upper middle third	Not stated	Not stated	Squamous-cell
Yes	"Large ulcerating lesion"; not annular	Distal third	None; irradiation	9 plus months	Squamous-cell, grade II
Yes	"Ulcerating and bleeding mass"; ? annular	Middle third	None; irradiation	10 months	Squamous-cell, grade III
Yes	Ulcerating growth; not annular	Middle third	Gastrostomy	15 days	Associated carcinoma of cardia; squamous-cell in type as was esophageal lesion
Yes	Not done	Middle third	Heller—cardiomyotomy	Less than 30 days	Squamous-cell carcinoma
Not done	Not done; tumor ⅞ths of circumference on post mortem	Distal third	Jejunostomy	7 days	Low-grade squamous-cell carcinoma
Yes	Ulcerating lesion	Middle third	Resection with esophagogastrostomy	Died of recurrence at 5 months	Epidermoid carcinoma, grade III; with metastases to upper gastric nodes

by the tumor to accomplish complete encirclement would be many times greater. Still, with complete encirclement, many patients exhibited an unrelenting dysphagia with loss of a previously intermittent character. This, or an advancing dysphagia after certain stability in its severity, we believe to be of diagnostic significance.

If food stasis or peptic regurgitation was a constant factor in mucosal neoplasia of the esophagus, one would expect to find carcinoma in the distal third of the esophagus. Actually, most cases showed malignant changes in the middle third of the esophagus or at the extremities of this segment.

One must expect that, unless an early diagnosis can be made, involvement by contiguous growth will occur before lateral submucosal growth will augment dysphagia or before intramural growth will manifest bleeding. The surgical cure of primary carcinoma of the esophagus is poor because of early invasion of adjacent vital structures; the cure rate of carcinoma in achalasia can be expected to be poorer.

Except in one instance, no direct surgical attack has been made in the cases reviewed. Sicher's[9] patient, who had one of the few lesions of the distal esophagus, was subjected to resection and esophagogastrostomy but survived only two days.

We subscribe to resectional surgery in these cases, if possible, on the same grounds that we subscribe to resectional surgery in "primary" esophageal carcinoma, particularly since irradiation offers nothing for the palliation of achalasia. The emotional reward from a patient who can swallow again buffers the moral uncertainty plaguing the surgeon who must do major palliative surgery.

We agree with Gore and Lam[2] that a right thoracotomy is desirable and utilized a technique similar to that described by DeBakey and Cooley.[11]

Fig. 1. Fig. 2.

Fig. 1.—Achalasia of five years' duration with recent unrelenting dysphagia. Note annular constriction with fluid level distally.

Fig. 2.—Postoperative barium study. Stomach in right hemithorax, less dilatation of proximal esophagus.

SUMMARY

1. Detailed published cases of carcinoma of the esophagus complicating achalasia have been reviewed.

2. Bleeding or progressive unrelenting dysphagia in achalasia warrants a careful search for carcinoma.

3. The need for periodic esophagoscopy and esophagograms is again stressed.

Addendum: Since this paper was submitted for publication, we have noted an article by Klayman (M. I. Klayman, Ann. Int. Med., **43**: 33-44, 1955) describing two patients with long-standing cardiospasm whose subsequent carcinoma was proved by exfoliative cytology, the possibility being suspect in only one of these.

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A TECHNICAL AID IN DILATATION OF ESOPHAGEAL STRICTURE

BENEDICT R. WALSKE, M.D.*
LINCOLN, NEB.

DILATATION of a benign stricture of the esophagus is a simple procedure in most instances and is deserving of little comment. There is little disagreement as to the safest methods to be employed. Blind methods are unsafe and are not to be recommended. Dilatation under direct vision or over a taut-string guide will prevent the creation of a false passage and all its sequelae. I, personally, prefer the latter since the method is simple and dilatation can be extended to the full normal diameter as indicated.

The passage of a string with a small lead shot attached would, indeed, appear to be a simple solution to the problem. There are instances, however, where the stricture is extremely narrow and there is a definite shelf or ledge just above the opening where the lead shot may "hang up" and pass with extreme difficulty. It occurred to me that a segment of fine watch chain or charm bracelet chain might be substituted for the lead shot. This has proved to be a most effective means of passing the guide string when a lead shot fails to pass. Fig. 1 shows the watch chain and a length of string. Fig. 2 shows the chain at proper level for adequate anchorage of the guide string.

A survey of the available literature did not disclose a report of a similar method. The simple technical aid described here has been most useful in a number of instances and is recommended to facilitate the passage of a guide string in a difficult case.

From the Veterans Administration Hospital, Lincoln, Neb.
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*Chief, Surgical Services and Thoracic Surgical Section, VA Hospital, Lincoln, Neb.

Fig. 1.

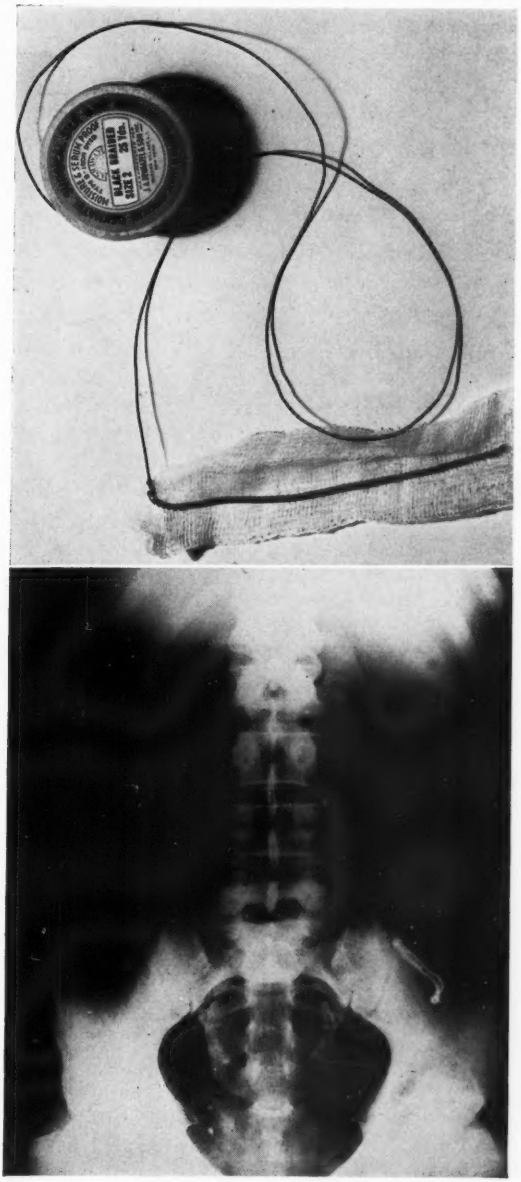

Fig. 2.

Fig. 1.—Heavy silk string attached to fine chain.
Fig. 2.—String and chain in proper position.

SURGICAL MANAGEMENT OF CONGENITAL TRACHEO-ESOPHAGEAL FISTULA AND ESOPHAGEAL ATRESIA

First Report of Patient Surviving Primary Operative Correction of Near Total Thoracic Esophageal Atresia

Ralph Berg, Jr., M.D.*
Spokane, Wash.

PRIOR to 1939, all forms of therapy for tracheo-esophageal fistula and esophageal atresia failed. No infant with esophageal atresia survived. Ladd[1] of Boston and Leven[2] of St. Paul, in 1939, independently, obtained survival in infants with esophageal atresia and distal fistula by closing the fistula and performing cervical esophagostomy and gastrostomy. In an infant treated by this method, the end result was compatible with life. However, the patient had a salivary fistula, had to be gastrostomy fed and the future held multiple-staged, often unsuccessful, operative procedures designed to establish some form of esophageal continuity. The expense was astronomical. Even in the few successful cases, the result was usually a malnourished child, unsightly in appearance.

Cameron Haight[3] of Ann Arbor, Michigan, in 1941, first established esophageal continuity successfully in a one-stage operative procedure by primary anastomosis of the proximal and distal segments. This can almost always be accomplished even in patients with nearly total thoracic esophageal atresias.

There are many combinations of atresia with and without fistula, but about 90 per cent are of the type of proximal atresia with distal tracheo-esophageal fistula. Early diagnosis of tracheo-esophageal fistula and esophageal atresia is essential to successful surgical therapy. One can hardly expect a reasonable surgical mortality if he is operating upon an infant five or six days old who is dehydrated, starved, and suffering from extensive chemical and bacterial pneumonia.

The cardinal symptoms are the baby's apparent excessive salivation and uniform respiratory distress when any attempt at feeding is made. Obviously, the blind proximal pouch results in saliva, water, or formula being aspirated or drooled from the mouth. The diagnosis is confirmed, when suspected, by passage of a soft rubber No. 10 or 12 F. catheter through the mouth into the pharynx and proximal esophagus. Of course, the catheter meets an obstruction in the esophagus. Contrast radiographic studies should be made for further confirmation and to exhibit the rare proximal segment fistulas. The presence or absence of gas shadows in the stomach and small bowel is observed. The degree of pneumonitis, as evidenced by pulmonary infiltration, is also estimated.

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*Address: 1003 West 32nd Avenue, Spokane 42, Wash.

About 25 per cent of these infants are less than 5 pounds in weight.[4] Those untreated die in approximately seven to fourteen days of dehydration, starvation, and pneumonia. The gastric secretion in the first few hours of life is highly acid, and the chemical pneumonia it produces on gaining entrance to the tracheo-bronchial tree through the distal fistula is added to that pneumonia already present from aspirated saliva and other liquids. Most of these babies have no other important associated anomaly. Those that do, have congenital heart disease, imperforate anus, and intestinal atresias and stenoses occurring in that order of frequency.[4]

Immediate operation should be performed except in those patients in whom a delay in diagnosis occurred. Appropriate time should be expended to correct ketosis, dehydration, and pulmonary infection by parental administration of fluid, glucose, vitamins, and antibiotic.

The babies are placed in Fowler's position to minimize regurgitation of acid gastric juice into the trachea. Periodic suctioning from the oropharynx prevents aspiration of mucus. Penicillin and streptomycin are the antibiotics we use by choice. Fluids and blood are administered via a polyethylene tube inserted into the left anterior malleolar vein. In my opinion, the anesthesia of choice for these infants is Pentothal sodium, occasionally supplemented with curare administered through the polyethylene tube inserted into the left anterior malleolar vein, with ventilation maintained by an endotracheal tube. Such controlled respiration makes possible a rapid, precise transpleural operation which may be completed in as little as one to one and one-half hours. To date we have not had reason to regret the use of the endotracheal tube.

The retropleural operative approach has long been abandoned by the author since it affords poor exposure and its deficiencies outweigh the advantages. The child is placed in the right chest position. The thorax is entered through the bed of the resected fourth rib (Fig. 1). The mediastinal pleura is entered by dividing the azygos vein. Pleural flaps are carefully preserved for later closure. Just inferior to the azygos vein, the bifurcation of the trachea is easily identified, and the fistulous communication with the distal esophagus dissected free. Care should be taken to avoid injury to the right vagus nerve* which is organized into one or two large trunks and courses obliquely on the lateral aspect of the esophagus and bears little resemblance to the nerve as seen in older subjects. This distal esophagus should be mobilized to the hiatus of the diaphragm; the blood supply enters the esophagus from the medial aspect and is easily preserved. Then the fistulous connection with the trachea is divided near the membranous portion and closed very adequately with a single suture ligature (Fig. 2). By dissecting the membranous portion of the trachea cephalad, the proximal pouch is usually very easily identified as a bulge in the mediastinum. Tenting this structure by a transoral catheter to aid identification is not necessary. The proximal pouch is freed with great care from the posterior membranous portion of the trachea

*In my experience, postoperative apparent pyloric or duodenal obstruction should be considered due to another lesion such as diaphragm, atresia, or pyloric sphincter hypertrophy, rather than loss of gastric tone and pylorospasm due to operative vagal injury. Valuable time may be lost in this erroneous concept (see Case 2).

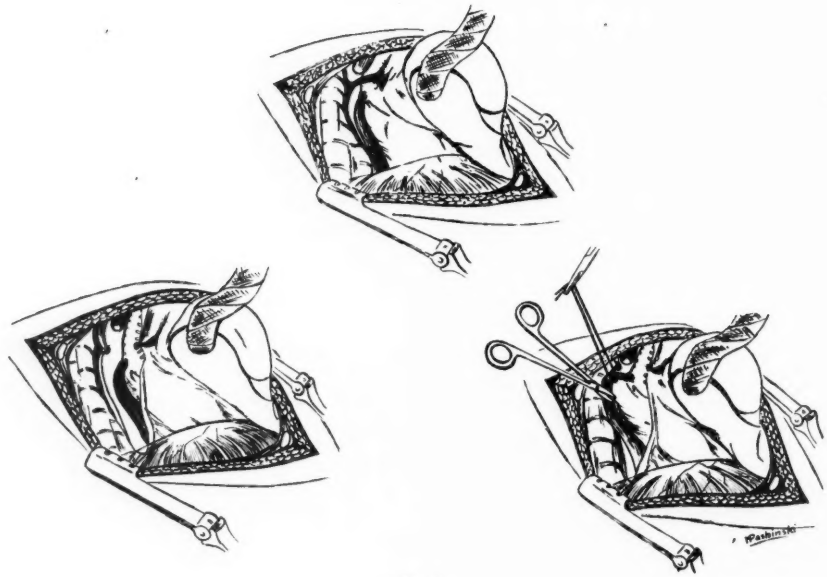

Fig. 1.

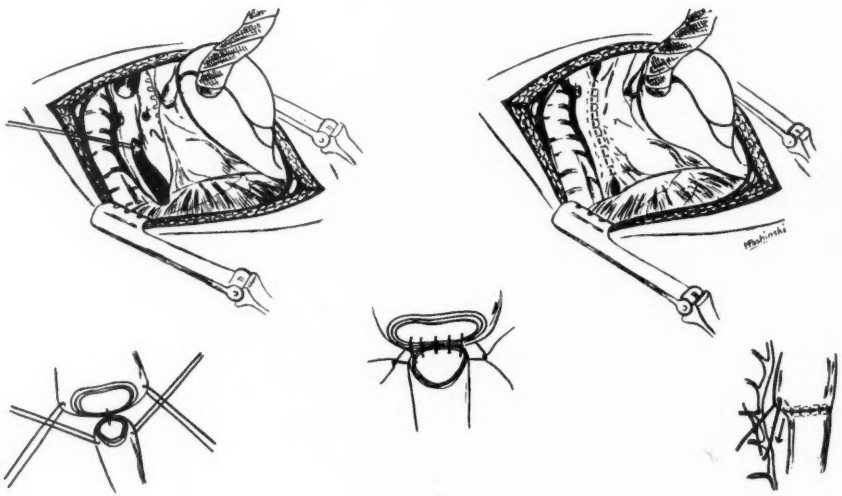

Fig. 2.

since an occasional second fistula may occur at that level. The proximal pouch should be then mobilized far into the neck by blunt and sharp dissection. Significant bleeding is seldom encountered. Devascularization of this pouch has not been a problem.

This will give satisfactory length for a primary anastomosis, which is made in a single layer with interrupted No. 5-0 silk sutures (Fig. 2). The anastomosis should be most meticulously fashioned as this is a critical step in the whole procedure. The proximal and distal ends should be handled only by means of stay sutures and not by tissue forceps. In my opinion, time is wasted on an inferior result when one performs a two-layer anastomosis. Furthermore, the excision of a button of tissue from the proximal segment seems to serve no useful purpose. This often inadvertently affords too large a proximal lumen and may sacrifice much needed length. It fosters mucosa muscularis separation which makes a precise one-layer anastomosis difficult.

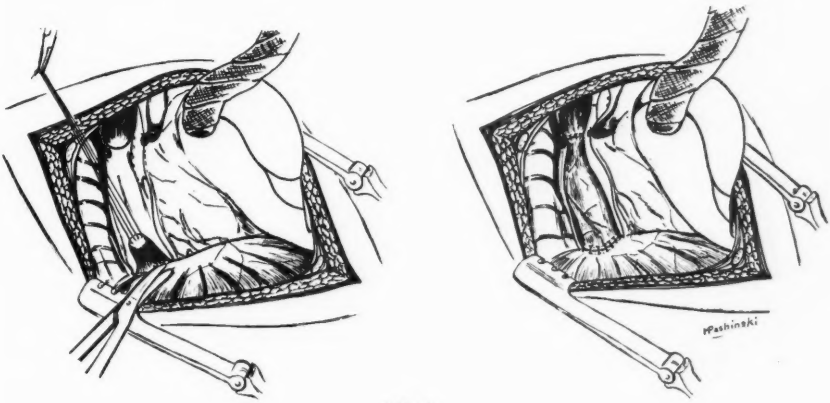

Fig. 3.

Suture over an indwelling tube has not been found necessary and results in lost time and increased contamination. After completion of the anastomosis, tension may be considerably relieved by suturing the proximal pouch to the anterior vertebral fascia (Fig. 2). The mediastinal pleural flaps are closed over the anastomosis with a running No. 4-0 plain catgut suture.

The presence of a distal fistula usually ensures a sufficient distal esophageal length for successful primary anastomosis. Large lengths of atresia without fistula may necessitate partial or nearly complete intrathoracic placement of the stomach to accomplish union (Fig. 3).

In my experience, with 1 case of nearly total thoracic esophageal atresia, the following observations were made:

1. A second incision in the rib cage through the bed of the eighth rib was necessary for exposure.

2. After incision of the pleura around the esophageal hiatus, it was easily enlarged to receive the stomach by separating the crural limbs of the opening.

3. Two factors interfered with mobilization; (a) the left gastric vessels which were divided, and (b) the attachments to the spleen on the left side.

Sufficient length was obtained for anastomosis as previously described with gratifying results (Patient C. G.). The wound was closed with interrupted fine catgut sutures, except the skin, and the pleural space drained by a catheter attached to a water seal. Following the surgery and blood replacement, the intravenous polyethylene cannula should be removed by the operator before the patient leaves the operating room. More of these infants have died in the past from overhydration in the postoperative period than from any other single postoperative factor. Postoperative pulmonary edema is usually the result of overhydration rather than any pulmonary injury incident to the anesthesia. These children require very little fluid in the immediate postoperative period. They should be in the oxygen-enriched humidified atmosphere of an incubator; a Stamm type gastrostomy is established in 24 to 48 hours following the thoracotomy. The alternate methods of early feeding without gastrostomy by use of an indwelling esophageal polyethylene tube have resulted in higher morbidity and mortality than gastrostomy. All postoperative fluid needs are well met by subcutaneous fluid until the gastrostomy is established. Penicillin, streptomycin, and vitamin K are routinely given throughout the postoperative period. The infant should be constantly attended with frequent aspirations of the pharyngeal secretions to prevent respiratory complications. If the esophageal anastomosis is under some tension, placing pillows and pads to produce anteflexion of the neck and trunk will relieve tension as flexing a bow loosens its string.[7] Oral feedings are begun on the sixth to eighth day, after satisfactory conditions are demonstrated by a Lipiodol swallow. The gastrostomy tube should be removed only when oral feedings have been well taken for at least one week and radiographs demonstrate an adequate esophageal lumen with no threat of stricture.

Complications which occur in the postoperative period are mediastinitis, pneumonitis, and suture-line leak. The later development of stricture is often due to prior leakage and mediastinitis. Most strictures are well treated by one dilatation. A few may require multiple instrumentations.

Dilatation of postoperative strictures should be done by the operating surgeon. Hubbard and Leven's[6] method of utilizing gum webbing type dilators passed after an attached filiform seems most satisfactory. The willowy filiform may be considerably stiffened by freezing with solid carbon dioxide just prior to use. The operator need allow only a few seconds to elapse after the filiform passes the stricture until it beomes warmed and sufficiently pliable to allow safe dilatation by the dilator which follows. Dilatation progressively to a No. 16 F. catheter or larger is done depending on the "feel." Stopping short of extensive mucosal tear, as indicated by bleeding, should lessen the tendency to restricture.

CASE REPORTS

CASE 1.—J. C. R. was a 7 pound, 7 ounce, male infant born on Jan. 29, 1953. Eighteen hours after birth, a diagnosis of tracheo-esophageal fistula was made by Dr. T. H. Shepherd. Radiographs demonstrated that the orally inserted catheter was arrested at the level of the second thoracic vertebra. There were right upper lobe pneumonitis and atelectasis with mediastinal shift to the right. A normal amount of gas was demonstrated in the stomach, small bowel, and large bowel in the pelvis. Laboratory data were a count of 5.7 million red cells with a hemoglobin of 17 Gm., a count of 12,600 white cells with a left shift. Albuminuria to a degree of 3 plus was found.

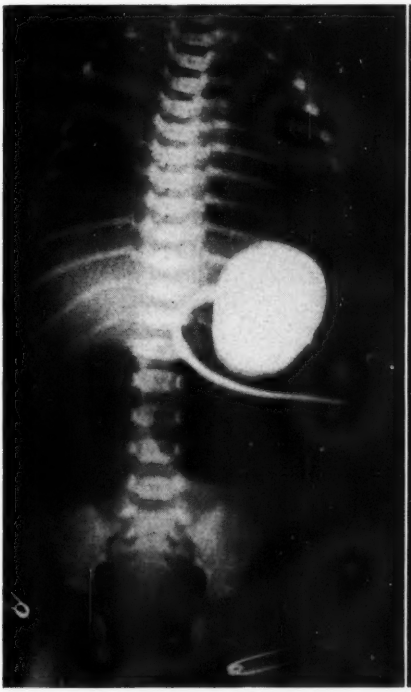

Fig. 4.

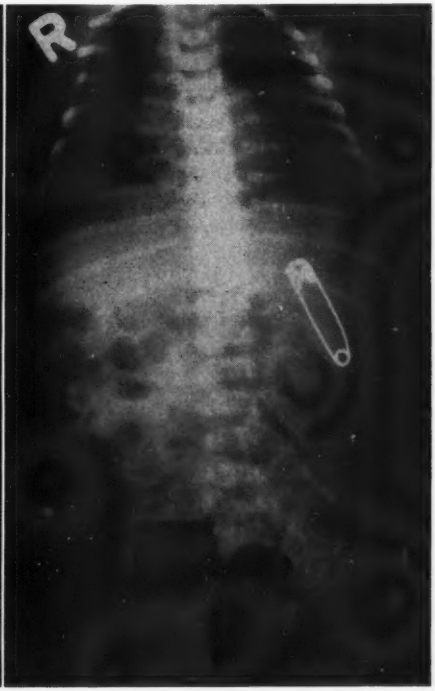

Fig. 5.

Fig. 4.—Case 1. (J.C.R.) Lipiodol has been instilled into the gastrostomy tube. The stomach, first, second, and third portion of the duodenum are greatly distended. There is no gas in the lower intestine. The major portion of the right fourth rib has been excised. The right lung is well expanded. The previously incomplete duodenal obstruction is now complete.

Fig. 5.—Case 1. Radiograph made March 16, 1953, the day before the child was discharged from the hospital. Excellent postoperative conditions exist in the thorax and the gastrointestinal gas shadows are within normal limits.

Twenty-three hours after birth, under intravenous Pentothal sodium and curare anesthesia and endotracheal control of respiration administered by Dr. S. T. Hubbard, the chest was entered through a right transpleural approach. A surgical correction of a tracheo-esophageal fistula with division and ligation of the tracheal portion of the fistula and anastomosis of the proximal and distal segments was performed. The pleural space was drained by a No. 10 F. catheter attached to a water seal. The operation was well tolerated. A few days later, a Stamm type gastrostomy was done.

Fig. 6.—Case 1. The Lipiodol esophagram made the day of discharge demonstrates an adequate esophageal lumen with narrowing at the site of anastomosis. There has been no dysphagia since operative correction of the lesion.

Fig. 7.—Case 1. (J.C.R.) The appearance of the child at 17 months of age.

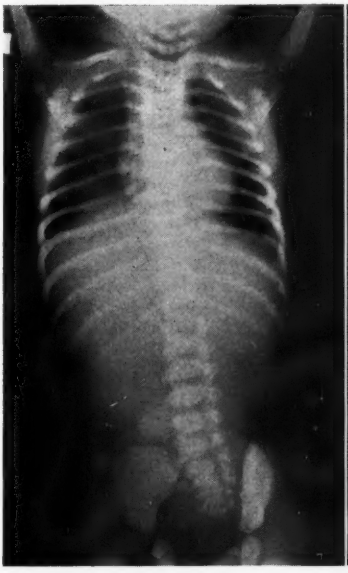

Fig. 8.

Fig. 9.

Fig. 8.—Case 2. (C.G.) This radiograph, taken at the age of 36 hours, demonstrates infiltration in the right upper lobe of the lung and absence of any intestinal gas shadows.

Fig. 9.—Case 2. (C.G.) A radiograph of a Lipiodol swallow demonstrates the esophagogastric anastomosis and intrathoracic position of the stomach. Since only a length of stomach necessary to accomplish anastomosis was placed in the chest, encroachment upon the lung with reduction of vital capacity is not a problem.

Fig. 10.—Case 2. (C.G.) This infant weighed 15 pounds at the age of 11 months, and is now well at the age of 20 months. Three esophagoscopic examinations with removal of foreign bodies and dilation of an esophagogastric stricture have been necessary. Her nutrition is excellent since she eats a normal diet. To my knowledge, this is the first reported case of the survival of a patient born with a nearly total thoracic esophageal atresia which was treated by primary esophagogastrostomy.

Although the child passed stools containing formula, it became apparent that a partial duodenal obstruction had become complete (Fig. 4). On Feb. 10, 1953, a duodenal diaphragm (with an eccentrically placed lumen, 3 mm. in diameter) was excised from the fourth portion of the duodenum. The child recovered and was discharged on March 17, 1953, in excellent condition (Figs. 5 and 6).

He is now 2 years and 5 months old and is, apparently, normal in every respect. There are no esophageal symptoms (Fig. 7).

CASE 2.—C. G. was a 6 pound, 12½ ounce, female infant, 19½ inches long when born on Sept. 30, 1953. A diagnosis of esophageal atresia was made by Dr. W. Lockwood. The child was 36 hours old at the time of diagnosis. Operation was performed at the age of about 40 hours. A right transpleural approach was utilized; the procedure being essentially that illustrated in Fig. 3. Anesthesia was administered by Dr. S. T. Hubbard and consisted of Pentothal sodium and curare with ventilation maintained by endotracheal tube. Anastomosis was fashioned over an indwelling polyethylene tube since the stomach had to be mobilized into the chest to provide gastroesophageal continuity, thus making gastrostomy undesirable. The patient's postoperative course was surprisingly uneventful. The Levin tube was removed on the fourteenth postoperative day and oral feedings were started on the same day. She was discharged on the twenty-eighth postoperative day.

COMMENT

It would seem logical to assume that almost all esophageal atresias could be corrected by a one-stage surgical procedure.

SUMMARY

1. In the past decade and one-half, tracheo-esophageal fistula and esophageal atresia has passed from a uniformly fatal lesion to one successfully treated surgically in a high percentage of the patients. A recovery rate of about 85 per cent is expected in those infants in whom prematurity is not a factor.

2. The report of a patient having survived surgical correction of a nearly total thoracic esophageal atresia has been presented, as well as one of a patient who survived operative correction of both tracheo-esophageal fistula and atresia and excision of a duodenal diaphragm which obstructed the fourth portion of the duodenum.

Excellent assistance in postoperative care of these patients by Drs. W. Lockwood, R. Heskett, and W. Davis is gratefully acknowledged.

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ARTERIALIZATION OF THE CORONARY SINUS IN OCCLUSIVE CORONARY ARTERY DISEASE

IV. Coronary Flow in Dogs With Aorticocoronary Sinus Anastomosis of Twelve Months' Duration

Alvin A. Bakst, M.D.,* and Charles P. Bailey, M.D.
Philadelphia, Pa.

THIS is a continuation of previous studies[1-3] undertaken to investigate the physiologic changes which occur in the coronary circulation following chronic arterialization of the coronary sinus for a period of twelve months.

During the course of our original experiments on the physiology of the coronary circulation in the sinus arterialized hearts of four to eight weeks' duration, we noted a definite protection afforded to the heart against ventricular fibrillation following the acute occlusion of the circumflex branch of the left coronary artery. However, in our subsequent experiments on hearts in which the coronary sinus had been arterialized for six months, we noted a significant loss of this protection.

It was obviously essential to study a series of animals in which the coronary sinus had been arterialized for a prolonged period of time, to determine whether there is a prolonged protection to the heart against the occlusion of a major coronary artery.

Pratt,[4] and Roberts and co-workers[5] originally described the concept of revascularizing the heart in occlusive coronary artery disease by the retrograde perfusion of the myocardium through the coronary sinus. Beck and his associates[6] have developed this procedure to its present two-stage technique. The procedure utilizes the placement of a free vein graft between the aorta and the coronary sinus at a first stage, followed by the subsequent partial ligation of the coronary sinus to 2 to 3 mm. at a second stage one month later.

Previously, Hahn and Beck[7] have stated that arterialization of the coronary sinus protected the heart against ventricular fibrillation following the ligation of a major coronary artery. This protection consisted of a reduction in both the mortality rate and the size of the infarct. Eckstein and co-workers[9, 10] have also described a protection afforded to the acutely arterialized heart following circumflex coronary artery ligation.

We, too, noted this protection to the heart in our original experiments on dogs in which the coronary sinus had been arterialized for four to eight

From the Department of Thoracic Surgery and the Brith Sholem Cardio-Pulmonary Laboratory, Hahnemann Medical College and Hospital, and the Bailey Thoracic Clinic, Philadelphia, Pa.

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*Present Address: The Jewish Hospital of Brooklyn, 555 Prospect Place, Brooklyn 38, N. Y.

weeks. However, because we could not substantiate this protection in animals in which the coronary sinus had been arterialized for six months, it became necessary to study a series of animals in which the coronary sinus had been arterialized for a truly prolonged period. This study, therefore, comprises a series of animals in which arterialization of the coronary sinus had been performed, and in which the animals were permitted to live a normal kennel existence for a period of one year prior to the institution of the physiologic experiments.

METHOD

Mongrel dogs, whose weight varied between 15 and 18 kilograms, were first prepared using the two-stage operation developed by Beck and his associates. At a first stage, through a left-sided approach, an external jugular vein graft was placed between the aorta and the coronary sinus. Approximately one month later, using a right-sided approach, the coronary sinus was partially ligated over a probe to 2 to 3 mm. Twelve months following the second stage, the physiologic data were obtained. At this time the animals were anesthetized with intravenous pentobarbital sodium, after which artificial respiration was maintained using a pneophore respirator attached to an endotracheal tube with inflated rubber cuff, to which intermittent positive pressure was supplied. The left chest was entered through the fourth intercostal space, and all pleural and pericardial adhesions were freed. The vein graft was inspected and only those animals in which it was patent were selected. An umbilical tape was placed around the graft to facilitate its occlusion at the appropriate moment during the experiment. A needle-tipped polythene catheter attached to a water manometer was inserted into the coronary sinus, through which the coronary sinus pressure and a blood sample for the determination of oxygen content were obtained. The graft was occluded for a period of two minutes, after which the experiment was repeated. The circumflex branch of the left coronary artery was next dissected to its origin and ligated. An incision was made in the wall of the artery, distal to the ligature, through which it was cannulated. A sample of retrograde circumflex coronary arterial blood was collected, simultaneous samples being obtained from the aorta and the pulmonary artery for the determination of oxygen content and saturation data. The minute volume of retrograde blood flow was recorded. At this point, the vein graft was occluded and the experiment was repeated. The peripheral segment of the circumflex coronary artery was permitted to bleed freely for a period of two minutes during which time the retrograde flow volumes were recorded, after which blood samples were collected for determination of oxygen content. This order for the collection of data was decided upon since, in our previous experiments, we noted that the retrograde blood oxygen content gradually increased over a two-minute period before it stabilized.

RESULTS

A. *Retrograde Blood Flow From the Peripheral Segment of the Ligated and Transected Circumflex Branch of the Left Coronary Artery.*—The results

are shown in Table I. With the graft open, retrograde blood flow ranged from 1.0 to 12.0 c.c. per minute, with an average of 6.0 c.c. per minute.

Following the occlusion of the aorticocoronary sinus vein graft, the retrograde circumflex artery blood flow ranged from 1.0 to 12.0 c.c. per minute, with an average of 6.6 c.c. per minute. Essentially, the retrograde blood flows remained unchanged in 4 animals and rose slightly in Dogs 2 and 6.

B. *Oxygen Content of the Retrograde Blood Collected From the Peripheral Segment of the Transected Circumflex Artery.*—The results are shown in Table I. The values for the oxygen content of this retrograde blood ranged from 12.9 to 18.7 volumes per cent with an average of 16.3 volumes per cent. Concomitantly, drawn samples of aortic blood ranged from 16.3 to 21.2 volumes per cent with an average of 19.0 volumes per cent.

On careful analysis of these data, it may be observed that the animals, in reality, divide themselves into two groups. In Dogs 2, 4, 5, and 6, the retrograde circumflex blood oxygen content closely approximates that of aortic blood, and is therefore arterial in nature. In Dogs 1 and 3, the retrograde circumflex blood oxygen contents are lower than those of the aortic blood, but are definitely higher than those of the pulmonary artery. They therefore represent a mixture between arterial and a small amount of venous blood. In none of these animals was the retrograde blood oxygen content within the range of the normal coronary sinus blood, as was originally found in the dogs in which true retroperfusion of the coronary sinus existed after one to two months of arterialization. Subsequent to the occlusion of the vein graft, the retrograde blood oxygen content rose farther toward that of aortic blood. These values ranged from 14.6 to 21.2 volumes per cent, and averaged 18.1 volumes per cent. In all the animals, there was a definite rise in the oxygen content of this back flow blood toward the value of the concomitantly drawn sample of aortic blood.

C. *Peripheral Pressure of the Distal Segment of the Transected Circumflex Coronary Artery.*—The results are shown in Table I. Peripheral coronary arterial pressures ranged from 16 cm. of water to 35 cm. of water and averaged 27.5 cm. of water.

Following the occlusion of the aorticocoronary sinus vein graft, the pressure within the peripheral segment of the circumflex coronary artery ranged from 16 to 55 cm. of water, and averaged 34.5 cm. of water.

D. *Mean Coronary Sinus Pressure and Blood Oxygen Content.*—The results are shown in Table I. With the graft open, the mean coronary sinus pressure ranged from 90 to 120 cm. of water and averaged 104 cm. of water. The oxygen content of the arterialized coronary sinus blood ranged from 15.7 to 19.2 volumes per cent with an average of 17.8 volumes per cent. Following the occlusion of the aorticocoronary sinus vein graft, the pressure within the coronary sinus rapidly fell to normal. The pressures now ranged from 4.0 to 6.0 cm. of water and averaged 4.8 cm. of water. The coronary sinus blood oxygen content now ranged from 1.0 to 10.3 volumes per cent with an average of 7.1 volumes per cent.

TABLE I. ARTERIALIZATION OF THE CORONARY SINUS—CORONARY FLOW, AND OXYGEN CONTENT OF BLOOD OBTAINED FROM THE PERIPHERAL SEGMENT OF THE CIRCUMFLEX CORONARY ARTERY—DOGS OF TWELVE MONTHS' DURATION

	CIRCUMFLEX CORONARY ARTERY						AORTIC BLOOD				CORONARY SINUS				PULMONARY ARTERY
	GRAFT OPEN			GRAFT CLOSED							GRAFT OPEN		GRAFT CLOSED		
DOG NUMBER	FLOW (C.C./MIN.)	O_2 CONTENT (VOL. %)	PRESSURE (CM. H_2O)	FLOW (C.C./MIN.)	O_2 CONTENT (VOL. %)	PRESSURE (CM. H_2O)	CONTENT	CAPACITY	SATURATION	PRESSURE	PRESSURE (CM. H_2O)	O_2 CONTENT (VOL. %)	PRESSURE (CM. H_2O)	O_2 CONTENT (VOL. %)	O_2 CONTENT (VOL. %)
1	1.2	12.9	26	1.2	16.8	55	16.3	16.5	98.8	95	90	17.7	5.6	10.1	10.3
2	9.0	18.7	27	11.0	21.2	39	21.2	19.7	107.1	100	100	19.2	4.0	9.9	13.3
3	1.0	13.3	16	1.0	14.6	16	19.8	18.5	107.0	108	106	17.6	4.4	1.0	9.0
4	7.8	18.7	40	7.5	18.2	37	18.7	19.2	97.3	110	110	15.7	6.0		11.0
5	12.0	17.8	35	12.0	20.6	33	20.6	20.4	100.0	100	100	18.9	5.0	11.6	13.7
6	5.0	16.4	21	7.0	17.5	27	17.1	18.1	94.5	120	120	17.7	4.0	10.3	12.3
Average	6.0	16.3	27.5	6.6	18.1	34.5	19.0	18.7	100.8	105	104	17.8	4.8	7.1	11.6

E. *Mortality Rate Following Circumflex Coronary Artery Ligation in Animals With Arterialization of the Coronary Sinus of Twelve Months' Duration.*—Since we desired to study all specimens pathologically, we selected an arbitrary survival time of four hours, subsequent to the ligation of the circumflex coronary artery, as the criterion for a comparative mortality report. Those animals which survived for four hours, following the ligation of the circumflex coronary artery, were classified as survivals. We thus continue to utilize the same criteria for survival as was established in our previous experiments. In the present series, 2 dogs survived for the prescribed period of time prior to sacrifice. Four dogs died of ventricular fibrillation within one hour of the ligation of the circumflex artery. This provides a mortality rate of 66 per cent. The records of the survivors (Dogs 2 and 5) may be seen in Table I. They demonstrated the highest rates of retrograde circumflex arterial flow. These flow rates were unchanged following the occlusion of the graft which probably indicates the existence of a more effective coronary collateral circulation.

DISCUSSION

The present series of experiments on animals in which the coronary sinus had been arterialized for a period of twelve months concludes an investigation, that we have been conducting for the past several years, of the effects of arterialization of the coronary sinus on the coronary circulation. Since we have previously investigated the physiologic effects of arterialization of the coronary sinus on the coronary circulation of animals over shorter periods of time, it would be valuable to review these findings.

In a series of normal control animals, we have demonstrated a small quantity of intercoronary collateral flow, measuring approximately 1.7 c.c. per minute. This blood was invariably arterial in nature, which demonstrated its origin to be from the neighboring coronary arteries. This backflow therefore represented a measure of normal arterial coronary collateral flow.

In a series of animals in which the coronary sinus had been arterialized for four to eight weeks, these findings were significantly changed.[2] With the graft functioning, the retrograde circumflex arterial blood flow averaged 12.2 c.c. per minute, thus representing a significant increase over that found in the normal animals. Moreover, this back-blood which arose from the peripheral segment of the circumflex coronary artery differed from that of the normal animal since it was now markedly deoxygenated, the oxygen content being within the range of normal coronary sinus blood. The desaturation of this blood clearly demonstrated that there was a retroperfusion of the myocardial capillary bed, with extraction of oxygen by the myocardium. When the aorticocoronary sinus vein graft was occluded, the retrograde circumflex coronary arterial blood flow fell to 7.1 c.c. per minute and the oxygen content returned to normal. The arterial nature of this blood again signified that it had arisen from intercoronary anastomoses. This retrograde coronary arterial blood flow represented a fourfold increase above that normally expected, and therefore represented a significant increase in the intercoronary arterial anastomotic flow.

In the series of animals previously reported upon,[3] in which the coronary sinus had been arterialized for six months, the findings were significantly changed from those in the four- to eight-week group. Here, with the graft functioning, the retrograde coronary arterial blood flow averaged 8.3 c.c. per minute. This blood was highly oxygenated, the oxygen content being within the range of concomitantly drawn samples of aortic blood. The arterial nature of this blood pointed toward its origin from neighboring unligated arteries, thus demonstrating an increase in the intercoronary collateral flow. When the aorticocoronary sinus vein graft was occluded, the retrograde circumflex arterial blood flow again averaged 8.7 c.c. per minute. The oxygen content of the retrograde blood was again unchanged and remained arterial in nature. Strikingly, then, in those animals in which the coronary sinus had been arterialized for a period of six months, the retrograde circumflex coronary arterial blood flow could not be altered by the opening or the closing of the aorticocoronary sinus vein graft. Moreover, the oxygen content of this blood was also unaffected by the presence or absence of a functioning vein graft. In short, the significant finding in the series of animals in which the coronary sinus had been arterialized for a period of six months, was that there was no significant contribution to the retrograde circumflex arterial flow by the arterialized coronary sinus. We were able to demonstrate an increase in the intercoronary arterial anastomotic flow, but were unable to demonstrate any significant retroperfusion of the myocardial capillary bed.

The present investigation of the coronary circulation of animals in which the coronary sinus was arterialized for a prolonged period of one year's duration merely serves to reconfirm the findings that have been described in the six-month group of animals. With the graft open and functioning, the retrograde circumflex coronary arterial blood flow averaged 6 c.c. per minute. This blood was oxygenated and within the range of concomitantly drawn samples of aortic blood. There were a few animals in which the oxygen content of the retrograde blood was definitely lower than that of concomitantly drawn samples of aortic blood, but in all the dogs the oxygen content was significantly higher than that of concomitantly drawn samples of pulmonary arterial blood. The oxygen content of the retrograde coronary arterial blood averaged 16.3 volumes per cent as compared to an average aortic oxygen content of 19.0 volumes per cent, and a pulmonary arterial oxygen content of 11.6 volumes per cent. When the aorticocoronary sinus vein graft was clamped, the backflow from the peripheral segment of the circumflex coronary artery averaged 6.6 c.c. per minute, which is insignificantly changed from that observed with the graft open. The oxygen content of this retrograde blood averaged 18.1 volumes per cent; a 2 volume per cent increase over that found with the graft closed.

Significantly, in the present series of animals in which the coronary sinus was arterialized for a period of one year, the retrograde blood flow from the peripheral segment of a ligated and transected coronary artery, and oxygen

content of this blood are completely unaffected by the presence or absence of a functioning aorticocoronary sinus vein graft. The arterialized coronary sinus has apparently completely lost contact with the capillary bed.

It might be well to review the comparative mortality statistics compiled during the course of our series of experiments. Originally, when the circumflex branch of the left coronary artery was ligated in a group of normal animals, the mortality rate was 90 per cent. In a second group of animals in which previous nondescript cardiac procedures had been performed, coronary arterial ligation produced a mortality rate of only 66 per cent. In the series of animals in which the coronary sinus had been arterialized for one to two months, we were very impressed by an apparent protection afforded the heart against the onset of ventricular fibrillation following major coronary artery ligation. Here, the mortality rate fell to only 20 per cent. This impression was quickly dispelled by our subsequent experiments on animals in which the coronary sinus had been arterialized for six months. In this group, we were unable to ascertain any protection afforded to the heart against ventricular fibrillation by the presence of the aorticocoronary sinus vein graft. The mortality rate had risen to 66 per cent which resembled that found in the group in which only nondescript cardiac procedures had been performed. This fact was further substantiated by the results in the present twelve-month group of animals. Here, the mortality rate following circumflex coronary arterial ligation was again 66 per cent, which is again identical to that of the six-month group of animals and those having had nothing more than nondescript cardiac procedures. These findings again serve to confirm those recently reported by Eckstein and his associates,[10] as well as our own.[3] These workers noted that the retroperfusion of the myocardium, which existed in dogs in which the coronary sinus had been arterialized for four weeks, could no longer be demonstrated after three months. At the end of this time, they too noted that the retrograde circumflex coronary arterial blood again became arterial in nature, and therefore postulated its origin to be from the interarterial collateral circulation. They were able to demonstrate that blood which flowed in retrograde fashion from the peripheral segment of the ligated circumflex coronary artery, probably originated in the anterior descending coronary artery. They were unable to demonstrate any significant effect, by the arterialized coronary sinus, upon the blood arising from the peripheral segment of the ligated and transected coronary artery.

The retrograde circumflex arterial blood flows, as seen in Dogs 2 and 6, Table I, with the graft open and closed, bear comment. Here, the backflow increased following the occlusion of the aorticocoronary sinus vein graft. We have described this increase in retrograde flow following clamping of the graft in previous experiments on animals in which the coronary sinus had been arterialized for six months. Eckstein and his associates[9, 10] have similarly described this increase in several animals in which the sinus had been arterialized for periods longer than three months. At post-mortem examination of these hearts, we noted that the ostium of the coronary sinus had not

been reduced to 2 mm. Possibly, the orifice of 4 mm. which existed in these animals permitted an arteriovenous fistula of such magnitude that occlusion of the graft, by obviating the fistula, improved the coronary flow of the unligated anterior descending coronary artery, and thereby increased the interarterial collateral flow.

The long-term series of experiments reveal that the favorable effects produced by arterialization of the coronary sinus apparently regress over a prolonged period of time. We first observed this regression at the end of six months, and have definitely reconfirmed it at the end of a year.

Fig. 1.—Low-power magnification showing a major artery and vein. The artery is normal. The lumen of the vein is obliterated by severe intimal proliferation and thrombosis. Recanalization of the thrombosed lumen of the vein is evident.

The inability to demonstrate any retrograde perfusion of the myocardial capillary bed by physiologic means is further substantiated by the microscopic pathologic findings at the time of the post-mortem examination. There is a marked thickening of the coronary sinus itself due to intimal proliferation. Moreover, all of the major tributaries to the coronary sinus demonstrate a

severe degree of intimal proliferation. The lumina of these vessels are so markedly encroached upon, that they may be considered to have been completely occluded by this process (Figs. 1 and 2). Apparently, prolonged periods of coronary sinus hypertension produce a severe degree of intimal proliferation within the small and large veins which empty into it. This intimal proliferation effectively occludes these vessels and subsequently prevents any retroperfusion of the myocardial capillary bed.

Thus, since the original retroperfusion of the myocardial capillary bed is obviated after a period of time because of the high head of pressure within

Fig. 2.—High-power magnification of the vein reveals old thrombosis with organization and recanalization of the lumen.

the coronary sinus and its venous tributaries, it is apparent that the end result of the procedure is to stimulate the development of the intercoronary collaterals. It strikes us that, since the prolonged effect of arterialization of the coronary sinus is one of increasing the intercoronary arterial anastomotic flow, it may be possible to attain this result by a more simple procedure. We are therefore instituting an evaluation of simpler procedures designed to increase the intra-extracoronary collateral flow.

SUMMARY AND CONCLUSIONS

1. The retrograde blood flow and oxygen content of the blood from the peripheral segment of a ligated and transected circumflex artery have been determined in dogs in which the coronary sinus has been arterialized for a period of one year. These determinations have been performed with the graft open and closed.

2. With the graft open, coronary sinus pressure is within the range of that of the systemic circulation and the coronary sinus blood is of high oxygen content.

3. When the graft is occluded, the coronary sinus pressure and blood oxygen content revert to those of normal coronary sinus levels.

4. With the graft open, retrograde circumflex coronary arterial blood flow averages 6 c.c. per minute. This represents a threefold increase over that expected in the control animal, but is less than that previously found in the two- and six-month groups of animals. This blood is arterial in nature, approximating the oxygen content of aortic blood. The retrograde flow is probably due to an increase in the intercoronary arterial collateral circulation.

5. Occlusion of the aorticocoronary sinus vein graft does not significantly alter these findings. This indicates that after one year, the vein graft no longer contributes to the interarterial collateral circulation and is dispensable.

6. The mortality rate following circumflex coronary arterial ligation is 66 per cent in the one-year group of animals, which is unchanged from that previously reported in our six-month group of animals.

7. Retroperfusion of the myocardial capillary bed could not be demonstrated in this group of animals.

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MEASUREMENTS OF THE UNILATERAL RESIDUAL VOLUME*

ARTHUR A. SIEBENS, M.D.,** MELVIN M. NEWMAN, M.D.,*** RALPH E. SMITH, M.D.,**** AND LAWRENCE H. VAUGHAN, M.D.*****

WITH THE TECHNICAL ASSISTANCE OF BETTY L. VESTAL

ST. ALBANS, N. Y.

INTRODUCTION

IT HAS been among our objectives over the past three years to evaluate the effects of pulmonary resection.[1-3] Included in the studies has been the measurements of the total capacity of the lungs. Because such data present an inaccurate description of the effects of procedures affecting the two lungs unequally, we have sought to measure the total capacity of each lung individually in association with bronchospirometry.

References to similar attempts at measuring the unilateral residual volume are few. Björkman[4] has recorded the complete unilateral and bilateral volumetric data of 2 patients and Gaensler and Cugell[5] have listed such figures for 1 patient. Although a considerable experience with this procedure is indicated by the reports of Frenckner,[6] no figures are listed by which the unilateral technique can be compared with an established method for measuring bilateral volumes.

The present brief report includes the unilateral and bilateral total lung volumes of 9 patients. Six were selected because the pulmonary lesion was thought to affect the distribution or dimensions of the lung volumes minimally; their data are therefore considered to approximate the normal distribution. Three patients were chosen in order to illustrate the application of the technique where the conventional bilateral figures are of equivocal value.

CLINICAL MATERIAL

The 6 "control" patients (Table I) were men with pulmonary tuberculosis restricted either to the apex of the right or the left upper lobe or to the superior segment of the right lower lobe (Patient 0586). Each was a candidate for segmental resection at the time of study. The youngest patient was 18 and the oldest 39 years of age. The vital capacity was normal in each instance.

From the Cardiopulmonary Function Laboratory, United States Naval Hospital, St. Albans, New York.

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**Present address: Associate Professor of Physiology, State University of New York Medical Center, New York City.

***Present address: Assistant Professor of Surgery, State University of New York Medical Center, New York City.

****Present address: Assistant Professor of Medicine, University of Minnesota Medical School, Minneapolis, Minn.

*****Chief of Thoracic Surgical Section, Veterans Administration Hospital, East Orange, N. J.

TABLE I. UNILATERAL AND BILATERAL TOTAL LUNG VOLUMES IN SIX MEN WITH NORMAL UNILATERAL AND BILATERAL VITAL CAPACITIES

PATIENT NUMBER		0490	0500	0523	0586	0612	0627
AGE (YEARS)		23	23	39	18	34	21
B.S.A. (SQ. M.)		1.67	2.12	1.70	1.89	1.89	1.96
DIAGNOSIS		Mod. tbc LUL	Min. tbc RUL	Mod. tbc LUL	Min. tbc RLL	Min. tbc RUL	Mod. tbc LUL
VITAL CAPACITY	Rt. c.c.	2462	2774	2416	2850	2699	2662
	Lt. c.c.	1928	2328	2086	2508	2451	2143
	Sum. c.c.	4390	5102	4502	5358	5150	4805
	Bilat. c.c.	4832	5979	4622	5760	5294	5469
	Rt. %	56	54	54	53	52	55
	Lt. %	44	46	46	47	48	45
RESIDUAL VOLUME	Rt. c.c.	1198	1032	732	756	1418	528
	Lt. c.c.	1078	745	634	807	1298	302
	Sum. c.c.	2276	1777	1366	1563	2716	830
	Bilat. c.c.	2127	1660	1572	1490	2935	851
	Rt. %	53	58	54	48	52	64
	Lt. %	47	42	46	52	48	36
TOTAL LUNG CAPACITY	Rt. c.c.	3660	3806	3148	3606	4117	3190
	Lt. c.c.	3006	3073	2720	3315	3749	2445
	Sum. c.c.	6666	6879	5868	6921	7866	5635
	Bilat. c.c.	6959	7639	6194	7250	8229	6320
	Rt. %	55	55	54	52	52	57
	Lt. %	45	45	46	48	48	43
FUNCTIONAL RESIDUAL CAPACITY	Rt. c.c.	2094	1645	1268	1618	2160	1196
	Lt. c.c.	1729	1335	1045	1669	1995	948
	Sum. c.c.	3823	2980	2313	3287	4155	2144
	Bilat. c.c.	3537	3251	2528	3445	4495	2282
	Rt. %	55	55	55	49	52	56
	Lt. %	45	45	45	51	48	44
MIXING INDEX	Rt. % N_2	1.72	2.16	1.72	0.96	4.41	5.96
	Lt. % N_2	8.34	2.08	2.17	1.22	7.51	0.47
	Bilat. % N_2	2.52	2.42	0.52	0.60	2.13	0.72
$\left(\frac{\text{R.V.}}{\text{T.L.C.}}\right)$ ×100	Rt.	33	27	23	21	34	17
	Lt.	36	24	23	24	35	12
	Bilat.	31	22	25	21	36	13
$\left(\frac{\text{F.R.C.}}{\text{T.L.C.}}\right)$ × 100	Rt.	57	43	40	45	52	37
	Lt.	58	43	38	50	53	39
	Bilat.	51	43	41	48	55	36

Two of the other 3 patients (Table II, Figs. 1 and 2) had been treated surgically for pulmonary disease. Patient 0003 had had resections of seven bronchiectatic bronchopulmonary segments: the right middle lobe, the lingula, and the basilar segments of the left lower lobe. A postoperative empyema had resulted in fixation of the left hemidiaphragm (Fig. 1*A*). Patient 0866 had bilateral tuberculosis. A seven-rib thoracoplasty had been performed in an attempt to control cavitary disease on the left side (Fig. 2*A*). The ninth patient, Number 1026 (Table II, Fig. 3), also with bilateral pulmonary tuberculosis, was studied after spontaneously developing a right phrenic nerve paralysis (Fig. 3.1).

METHODS

The study of each patient included a measurement of the total capacity of both lungs as well as of each lung individually. In obtaining the conven-

TABLE II. UNILATERAL AND BILATERAL TOTAL LUNG VOLUMES IN THREE MEN WITH ABNORMALLY UNEQUAL UNILATERAL VOLUMES

PATIENT NUMBER		0003	0866	1026
AGE (YEARS)		46	39	29
B.S.A. (SQ. M.)		2.21	1.79	1.93
DIAGNOSIS		Bilat. resection bronchiectasis; Postop. empyema, Lt.	Bilat. tbc; Lt. thoracoplasty	Bilat. tbc; Apical disease; Rt. phrenic paralysis
VITAL CAPACITY	Rt. c.c.	2553	1566	654
	Lt. c.c.	1451	358	2187
	Sum. c.c.	4004	1924	2841
	Bilat. c.c.	4026	1944	3293
	Rt. %	64	81	23
	Lt. %	36	19	77
RESIDUAL VOLUME	Rt. c.c.	609	609	287
	Lt. c.c.	1484	353	688
	Sum. c.c.	2093	962	975
	Bilat. c.c.	2305	890	1005
	Rt. %	29	63	29
	Lt. %	71	37	71
TOTAL LUNG CAPACITY	Rt. c.c.	3162	2173	941
	Lt. c.c.	2935	711	2875
	Sum. c.c.	6097	2884	3816
	Bilat. c.c.	6331	2834	4298
	Rt. %	52	75	25
	Lt. %	48	25	75
FUNCTIONAL RESIDUAL CAPACITY	Rt. c.c.	1141	1108	437
	Lt. c.c.	1914	398	1004
	Sum. c.c.	3055	1506	1441
	Bilat. c.c.	3070	1458	1564
	Rt. %	37	74	30
	Lt. %	63	26	70
MIXING INDEX	Rt. % N_2	1.79	0.90	2.40
	Lt. % N_2	1.01	3.88	0.90
	Bilat. % N_2	2.61	1.88	1.01
$\left(\frac{\text{R.V.}}{\text{T.L.C.}}\right) \times 100$	Rt.	19	28	30
	Lt.	51	50	24
	Bilat.	36	31	23
$\left(\frac{\text{F.R.C.}}{\text{T.L.C.}}\right) \times 100$	Rt.	36	51	46
	Lt.	65	56	35
	Bilat.	48	51	36

tional bilateral figures, the functional residual capacity was measured twice, using a modification of the Darling[7] method. Duplicate analyses agreed within 10 per cent; the mean of these two values appears in the tables. The bilateral index of intrapulmonary mixing listed in the tables is similarly a mean of the nitrogen concentrations in the "alveolar" air samples delivered at the end of each seven-minute period of oxygen breathing. The vital capacity and its subdivisions were recorded in triplicate, the best of the three tracings being used in computing the residual volume and the total lung capacity.

Bronchospirometry was performed under 1 per cent Pontocaine local anesthesia after premedication with Nembutal, atropine, and morphine. The catheter (Carlens[8]) was introduced, usually over a flexible sound, into the upper larynx under mirror visualization. It was gently rotated through the

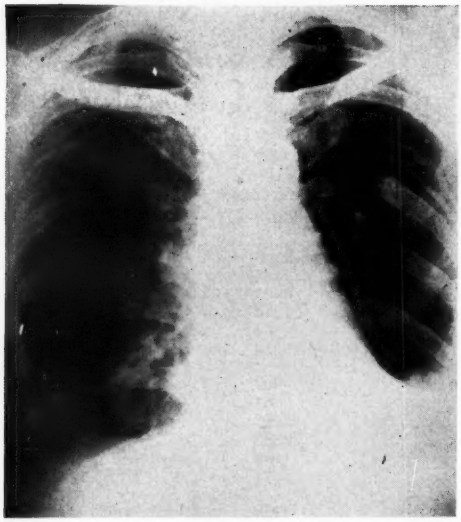

Fig. 1*A*.—Patient 0003. Bilateral resections for bronchiectasis. Postoperative chest roentgenogram. Note evidence of previous empyema on the left with pleural thickening and elevation of diaphragm. At fluoroscopy the left hemidiaphragm was found to move minimally on forced inspiration and expiration, the left rib cage was relatively fixed and the left lung field was more radiolucent than the right.

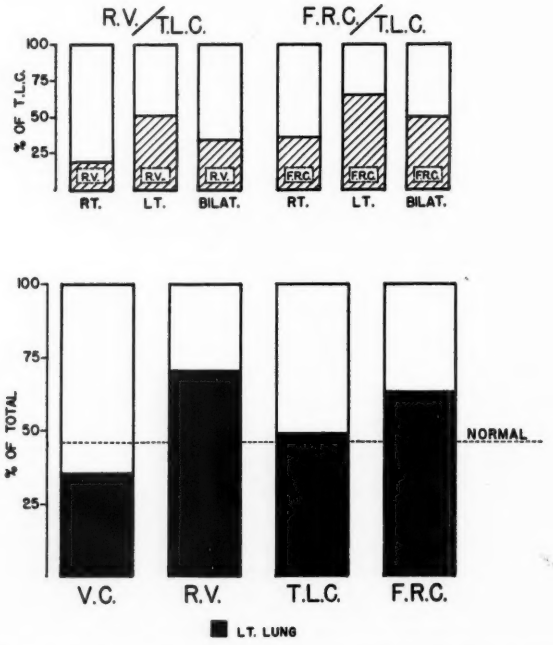

Fig. 1*B*.—Patient 0003. Unilateral and bilateral lung volumes and volumetric ratios. Note: (1) Although the total lung capacity is normally distributed, the other compartments are not: the vital capacity on the left is low but the residual volume on this side is high. (2) The ratio of the bilateral residual volume to the total lung capacity is normal in spite of gross abnormality of this ratio on the left.

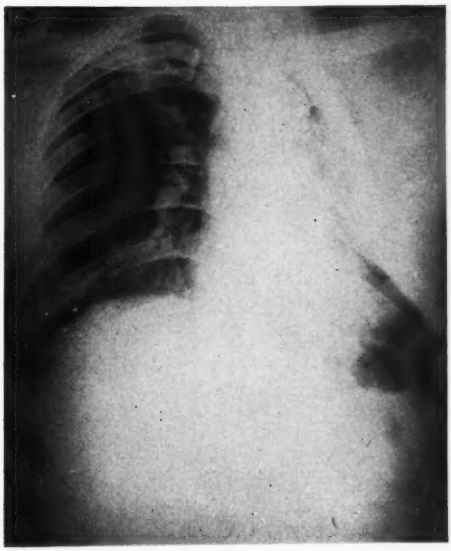

Fig. 2*A*.—Patient 0866. Bilateral pulmonary tuberculosis, left thoracoplasty.

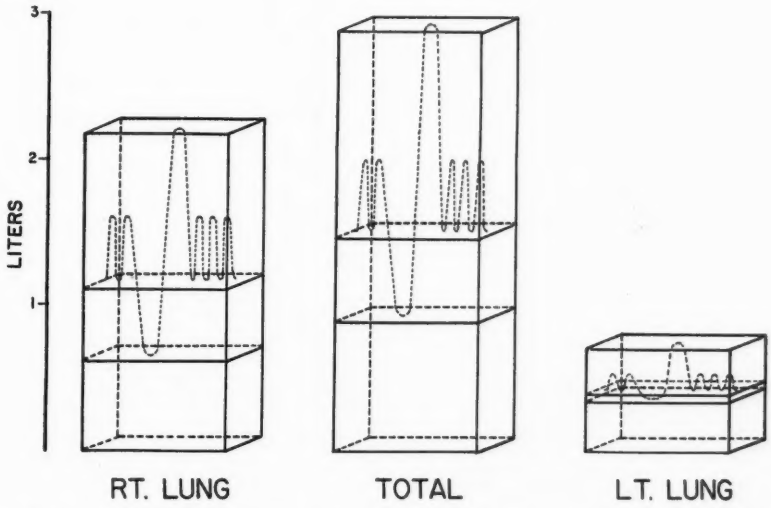

Fig. 2*B*.—Patient 0866. Unilateral and bilateral lung volumes. Note the magnitude of the differences in the distributions of the various volumetric compartments. These are identified by name in Fig. 3*B*.

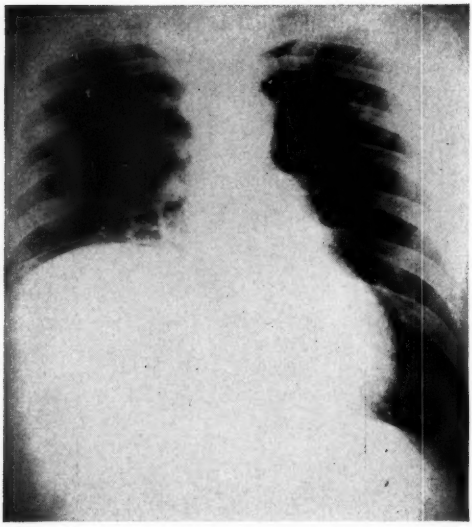

Fig. 3*A*.—Patient 1026. Bilateral pulmonary tuberculosis, spontaneous right phrenic nerve paralysis.

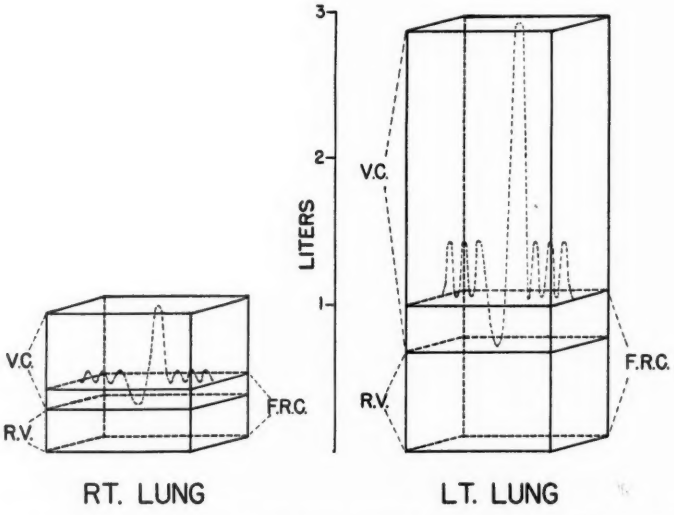

Fig. 3*B*.—Patient 1026. Unilateral lung volumes. Note the practically symmetrical reduction in the various subdivisions of the total capacity of the right lung. V.C.—vital capacity, R.V.—residual volume, F.R.C.—functional residual capacity.

glottis after removal of mirror and sound and was positioned with the assistance of a fluoroscope. Its spur was not tied down for passage into the trachea.

The two lumina of the catheter were initially coupled to a brochospirometer the bells of which were filled with room air. After a satisfactory three-minute tracing of oxygen consumption and tidal respiration had been recorded, the patient was instructed to deliver his expiratory reserve and, following restoration of the mid-position, his inspiratory capacity. The bronchospirometer was then uncoupled and each side of the catheter was fitted to a mobile unit for measuring the unilateral functional residual capacity. Each unit consisted of a vertically adjustable table on which lay a Douglas bag for collecting expired air. The coupling to the patient led into a valve which could be turned either into a circuit incorporating this bag with one-way valves and a source of oxygen or into an arm communicating optionally with the room or with an underwater trap for collecting the sample of "alveolar" air. A check valve prevented aspiration of the water in the trap. The patient was switched from room air into the oxygen circuit at the end of what was judged to be a normal expiration. The expired air was collected for nine minutes, during which time nitrogen was displaced from the alveolar trap circuit by flushing with oxygen. At the end of the nine minutes, the patient was instructed to inspire normally and hold his breath while the valves were turned to the alveolar traps. He then expired slowly and fully through the trap, the valve being turned to room air just prior to inspiration.

In most instances, it was possible as a final step to obtain a second bronchospirometric tracing as a check on the location of the catheter. When a change in position was indicated, the determination was discarded.

The volume of the catheter lumina, connecting tubes, and valves was 130 c.c. Since the anatomic dead space for which this volume was substituted approximates 150 c.c., no correction was included. The figure for nitrogen excretion incorporated in the equation for calculating the functional residual capacity was divided between the two lungs according to the distribution of the oxygen consumption.

Nitrogen analyses were performed in Van Slyke manometric equipment. All lung volumes were converted to body temperature, ambient pressure, saturated with water vapor (BTPS).

RESULTS (TABLES I AND II)

In these 9 patients, the sum of the *functional residual capacities* of the two lungs differed from the mean of the duplicate bilateral values by less than 10 per cent.* This level of agreement was found for the *residual volume* in all but 1 of the patients (No. 0523, 13.1 per cent), with respect to the *vital capacity* with three exceptions (No. 0500, 14.7 per cent; No. 0627, 12.1 per cent; and No. 1026, 13.7 per cent), and for the *total lung capacity* with two exceptions (No. 0627, 10.8 per cent; and No. 1026, 11.2 per cent). Percentile correspondence of the two techniques with respect to residual volume and func-

*Calculated by dividing the difference between bilateral and sum of unilateral volumes by the bilateral value.

tional residual capacity, therefore, fell well within the range found for vital capacity. Since the former two compartments are considerably smaller than the latter, the actual volumetric differences were considerably less.

Mean values for the distribution of the various compartments between the two lungs in the "normal" group (Table I) were nearly identical, being 54 per cent on the right and 46 per cent on the left except for the residual volume (55 per cent right, 45 per cent left.) Variations in the distribution of the residual volume ranged from 48 per cent to 64 per cent for the right lung. Corresponding figures for the functional residual capacity and vital capacity, respectively, were 49 per cent to 56 per cent and 52 per cent to 56 per cent.

In the other 3 patients (Table II) the distribution of the lung volumes differed markedly from the normal. Patient 0003 had 36 per cent of the vital capacity on the left but 71 per cent of the residual volume on this side (Fig. 1*B*). Corresponding figures for Patient 0866 were 19 per cent and 37 per cent, respectively (Fig. 2*B*). In Patient 1026, on the other hand, all compartments were proportionately reduced on the side of phrenic paralysis (Fig. 3*B*).

Among the six "normals," differences in excess of 4 per cent between a unilateral and the bilateral R.V./T.L.C. or F.R.C./T.L.C.* were found for 3 of the 12 lungs (No. 0490—left R.V./T.L.C., left and right F.R.C./T.L.C.; No. 0500—right R.V./T.L.C.). There were differences of this magnitude between the 2 lungs of the same individual in two instances (No. 0627—R.V./T.L.C.; No. 0586—F.R.C./T.L.C.). As would be expected, the variations between the 2 lungs and between 1 lung and the bilateral value were large in the 3 abnormal patients.

The mixing index was grossly different (more than 2 volumes per cent nitrogen) in four of eighteen comparisons between a unilateral and the mean of two bilateral values (Patients 0490, 0612, and 0627). The unilateral concentrations were usually somewhat higher than the bilateral for reasons which will be discussed.

DISCUSSION

Measuring the functional residual capacity of each lung individually by the method employed is time consuming and technically difficult. Eight nitrogen analyses, including unilateral and bilateral determinations, are needed for each complete study. The usual problems of satisfactory bronchospirometry, in particular the presence of secretions, are compounded by the added time required. The impediments to normal breathing created by the presence of the catheter result in a high incidence of poor checks between unilateral and bilateral functional residual capacities. Since a reasonable agreement by the two techniques is essential for evaluating a given unilateral analysis, data must be discarded frequently.

A number of factors are helpful in increasing the yield of acceptable studies. Primary among these are selecting cooperative patients with dry bronchial trees and developing a smooth, rapid routine for the various steps involved.

*R.V./T.L.C. = ratio of the residual volume to the total lung capacity; F.R.C./T.L.C. = ratio of the functional residual capacity to the total lung capacity.

Difficulties notwithstanding, however, it is evident that knowledge of the unilateral residual volume is occasionally essential to an intelligible analysis of volumetric status. The usual bilateral and unilateral measurements of Patient 0003, for example, fail to suggest the extent of the abnormality resulting from fixation of the left diaphragm in a depressed position. Assessing the effects of phrenic interruption, as in Patient 1026, and of various forms of unilateral collapse or resection therapy requires a knowledge of unilateral residual volume. It should be noted that the elevation of the right diaphragm and consequent low functional residual capacity for the right lung in Patient 1026 (Figs. 3*A* and 3*B*) is rarely seen following therapeutic phrenic nerve interruption, a procedure which in the absence of pneumoperitoneum usually fails to change the unilateral mid-position.[5, 9]

The limitations imposed by a knowledge only of the bilateral residual volume and the unilateral vital capacity, apparent in evaluating the effects of resection in Patient 0003, are also illustrated in the studies of Patient 0866 (Figs. 2*A* and 2*B*). The magnitude of the disproportion in the distribution of vital capacity (19 per cent) and residual volume (37 per cent) on the side of thoracoplasty would have been difficult to surmise in the latter without the unilateral figures. Although the very high proportion of the total lung capacity comprised by the residual volume in this patient's left lung (50 per cent) might be interpreted to indicate "overdistention," it is doubtful that this ratio has the same connotation in this instance that it usually carries where there is no deformation of the parietes and where there exists no gross alteration in the relationship between tissue mass and its enclosure.

It was observed in the description of results that the distribution of the residual volume varied more among the "normals" than did that of the other compartments. It should be noted that in the two instances in which departure from the mean was most pronounced (Patients 0586 and 0627), the residual volume on the side of the lesion was unusually low. A similar effect of relatively small, unilateral, parenchymal lesions on the ipsilateral residual volume was also found occasionally among patients selected for phrenic nerve interruption.[9]

One might anticipate that results with the bronchospirometric technique would be influenced by the resistance to respiration added by the Carlens tube.[10] Were this factor of major importance, the functional residual capacities measured by the unilateral technique should be higher than the bilateral figures.[11] Since such a difference exceeding 50 c.c. was found only once (Patient 0490, 286 c.c.), any additional resistance encountered by these 9 patients was probably not pronounced.

Early in the development of the unilateral technique, the concentration of nitrogen in "alveolar" air was frequently found to be abnormally high in comparison with the bilateral figure. In order to reduce the magnitude of this difference, the period of oxygen breathing for the unilateral procedure was extended to nine minutes as compared to the conventional seven. With this modification, concentrations below 2.5 per cent were found in thirteen of the eighteen unilateral analyses included in this report. Of the remaining five,

one (Patient 0490, left lung) was associated with a high bilateral figure, two were observed in a patient with an unusually large functional residual capacity (No. 0612) and a marginal bilateral index (2.13 per cent), one could be attributed to poor ventilation (17 per cent of total) on the side of thoracoplasty (Patient 0866), and in the final instance (No. 0627, right lung) the elevation could be explained on no reasonable basis other than contamination. The likelihood of a continuous air leak in the latter is excluded by the normal distribution of the functional residual capacity and the close correspondence of unilateral and bilateral figures. A small admixture of room air during delivery of the sample or inadequate flushing of the alveolar air trap are most likely to have been responsible.

The basic reason for differences in nitrogen emptying rates with the two techniques, however, is in all likelihood our failure to reduce the volume of tubing and valves between inspiratory and expiratory orifices on the unilateral oxygen circuits commensurately with the reduction in tidal volume due to unilateral respiration. In this circumstance, the ratio of dead space to tidal exchange for the unilateral procedure is in excess of the figure for the conventional technique. Accordingly, a smaller proportion of the gases escaping through the expiratory valve are alveolar in composition, tidal volumes of oxygen are poured into a relatively larger reservoir during inspiration, and nitrogen elimination by serial dilutions is retarded.

Finally, it should be observed that the above considerations, although reflecting on the interpretation of the unilateral alveolar index, do not introduce appreciable limitations with respect to the comparison of unilateral and bilateral functional residual capacities. Because the equation for computing the functional residual capacity is relatively insensitive to final nitrogen concentrations, differences between unilateral and bilateral indices, unless extreme, result in small differences in the measurement of this volumetric compartment.*

SUMMARY

The residual volume of each lung individually was measured in association with bronchospirometry. In each of 9 patients, the sum of the functional residual capacities of the two lungs differed from the mean of two bilateral measurements by less than 10 per cent; in 8 of the 9 a similar level of agreement was found for the residual volume. The degree of correspondence for these volumetric compartments compared favorably with those established for the vital capacity and the total lung capacity.

Among 6 patients with minimal or moderate pulmonary tuberculosis and normal vital capacities the mean values for the distribution of the various compartments between the two lungs were nearly identical (54 per cent right, 46 per cent left for the functional residual capacity, vital capacity, and total lung

*If the bilateral functional residual capacity were 3,000 c.c. and the mixing index 1 per cent, for example, a concentration as different as 5 per cent would result in a volume of 3,160 c.c. (error of 5.3 per cent).

capacity, and 55 per cent right, 45 per cent left for the residual volume). The extremes of variation around the mean were greatest with respect to the residual volume.

The advantage of knowing the unilateral residual volume was illustrated by application of the technique to 3 patients whose conventional bilateral measurements afforded an equivocal definition of volumetric status.

The bilateral studies and nitrogen analyses were performed by hospital corpsmen of the United States Navy trained in the Cardiopulmonary Laboratory of the U. S. Naval Hospital, St. Albans, New York. The interest and skill of the following are acknowledged with particular gratitude: John Briggs, Kenneth Gorr, Roy Johannessen, Joseph Malin, Richard Monsees, Earnest Picknally, David Prior, and Robert Rauf.

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MANAGEMENT OF THE POSTRESECTION SPACE IN TUBERCULOSIS

III. Role of Pre- and Postresection Thoracoplasty

John W. Bell, M.D.
Sunmount, N. Y.

THORACOPLASTY, for several decades the major surgical treatment of tuberculosis, has seemingly receded into the limbo of the past since 1950.[1] With the rapid evolution of thoracic surgery and the spectacular use of antituberculosis drugs, pulmonary resection has become the principal method of surgical treatment at the beginning of this fifty-year span.

Although the indications and results of collapse therapy have been clearly drawn, the majority of these operations were performed in the preantibiotic era.[2, 3] The successful application of various excisional techniques to all stages of tuberculosis has made it difficult to evaluate the role of thoracoplasty when associated with lung resection. Whether today's modification of the standard posterolateral thoracoplasty, the so-called apical tailoring thoracoplasty, is a useful adjunct in certain cases; or whether it is simply a carry over, and indeed should be discarded altogether, is not clear from current writing.

In accord with our study of the postresection space in tuberculosis, and various measures to avoid or treat such a space, we have reviewed the indications and results of pre- and postresection thoracoplasties, as performed at the Veterans Administration Hospital (Sunmount, New York), over a five-year period (1950 to 1955).

PRERESECTION THORACOPLASTY

During the period of transition (1940's), when thoracoplasty was gradually yielding to resection as the dominant surgical treatment, experienced thoracic surgeons believed that resection was safer for certain types of patients when they had had a previous thoracoplasty. Prior to the systematic application of prolonged, continuous chemotherapy, no methods other than major or minor collapse were available for the control of progressive, exudative disease. Patients with moderate or far-advanced tuberculosis, who showed no inherent resistance to their disease under conditions of maximum bed rest and improved nutrition, were treated with varieties of pulmonary collapse, often on an emergency basis.

The amazing success which specific antimicrobial agents have had in aiding the resolution of pneumonic or prenecrotic tuberculosis, has made the control of the acute case largely a medical, rather than surgical, problem. The average

From the Veterans Administration Hospital, Sunmount, N. Y.
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surgical candidate today may come to resection in the sputum-negative state, with bacilli on the last positive culture completely susceptible to the particular drugs being used.

A small group of patients, perhaps 10 per cent, even with the administration of several combinations of prolonged drug regimens, will not, after six to nine months, show permanent sputum conversion or cavity closure. In an effort to control these patients for ultimate resection, and following the doctrine of the thoracoplasty era, a number of preresection thoracoplasties were performed. Our specific indications were: patients with unequivocal upper lobe cavities and persistent or intermittently positive sputum in whom the greater part of the exudative element had cleared. Unfortunately, many of this group had bacilli resistant to one or two drugs and uncertain coverage with a third agent. The anticipation of pleural complications in this group, plus the desire to minimize them, led to a recommendation of preresection thoracoplasty. Further, it was hoped that a period of collapse might aid the stability of disease in these patients who had had only a moderate response to the drug program. A second consideration was more mechanical than chemotherapeutic. In this same group were patients with scattered middle and lower lung field lesions, associated with major upper lobe involvement. Fluoroscopy revealed a 2 to 3 plus trapping in the lower lobes, with various degrees of obstructive emphysema demonstrated in pulmonary function screening. The expectation was that such lower and middle lobes would exhibit delay in occupation of the apex following upper lobectomy or bisegmental excision. The prospect of a space problem was then a further reason for preresection thoracoplasty.

Our plan of preresection thoracoplasty has been the subperiosteal removal of the first 3 ribs, with a routine apicolysis down to or near the level of the azygos vein or aorta, respectively. This operation was carried out in 29 patients, without complications. The interval from thoracoplasty to resection varied from a minimum of three to eight weeks. In older patients, a longer interval was allowed for increased stability of the regenerating plate.

The extent to which a first-stage, 3-rib thoracoplasty reduces over-all function is variable.[4] The physiologic age of the patient, the amount and distribution of tuberculosis in the underlying lung, the association of chronic bronchopulmonary conditions, such as asthma, emphysema, and interstitial fibrosis, are among the factors which make the functional loss unpredictable on an individual basis. It is generally assumed that a restricted collapse, over a relatively destroyed upper lobe, will result in very little net change.

Kaltreider and co-workers[5] commented that thoracoplasty produces a greater reduction in the ventilatory capacity than is to be expected from the extent of lung tissue compressed. Cournand and associates,[6] in a study of 200 cases, found that an apical thoracoplasty (up to 5 ribs) decreased the maximal breathing effort up to 15 per cent of its preoperative value. In a similar study, Gaensler and co-workers[7] concluded that the maximal breathing capacity was decreased about 20 per cent when 5 or 6 ribs were removed. We have not carried out pulmonary function studies in patients who had a specific preresection thoracoplasty followed by resection. Since full physiologic recovery from

thoracoplasty cannot be expected in less than four months (Gaensler found that loss of function at two weeks was nearly twice what it was at six months), it was felt that this determination would be of little value.

In order to avoid the physiologic handicap of paradox following thoracoplasty, it is customary to wait for variable intervals to allow hardening or bony regeneration of this area. Because the adequate clearing of secretions is dependent on the coordinate effort of all intercostal musculature with the diaphragm, "the bellows mechanism," even a rigid thoracoplasty plate means the upper third of the hemithorax is adding little to the cough mechanism. Pain from a fifth rib level incision further splints the lateral chest wall. In the case of a previous two-stage thoracoplasty, the plate itself may have to be cut for some distance when mobilizing the upper lobe for resection.

Analysis of the cough mechanism in human beings have shown that it is a complex coordination of muscular effort. Contraction of the intrinsic and extrinsic muscles of respiration, while the diaphragm aids the tussive force of the lungs in expelling secretions, indicates the importance of synchronous action and elastic recoil in the thoracic cage and its contents.

Consideration of the above observations, plus the impression of how a patient with a preresection thoracoplasty behaves after resection, compared with the behavior of a patient who has an upper lobectomy without thoracoplasty suggests that because of the presence of this thoracoplasty we have made these patients unnecessary surgical cripples.

Radiologic observations (Figs. 1, 2, and 3), during the first two postoperative weeks, reveal that the underlying lung is not ventilating as well, is not filling the hemithorax as promptly, nor clearing the operative reaction as soon as in the nonthoracoplasty patient. Particularly is the older patient handicapped. General muscle weakness, obesity, higher incidence of chronic emphysema and bronchitis, rob the man over 45 years of age of the extra respiratory effort he needs so badly.

There have been no tuberculous complications incident to the preresection thoracoplasty itself. Because it is a safe procedure, of course, does not mean it is necessarily a wise one. Major complications have followed the resection itself, and are probably more related to the chemotherapeutic control and the presence of unsuspected endobronchial disease and resistant organisms. There is no evidence to suggest that any of these complications were in any way avoided; in fact, some may have been precipitated by the presence of a preresection or standard thoracoplasty.

Minor postresection "mechanical" complications are, however, definitely increased in the preresection thoracoplasty group. Almost without exception, they have had a more difficult convalescence. Intratracheal aspiration, bronchoscopy, intermittent oxygen, aerosol mixtures and bronchodilators are all required with greater frequency in these patients.

There is no way to reconcile the hazards of performing a thoracoplasty simultaneously with a resection, unless paradox can be eliminated either by an

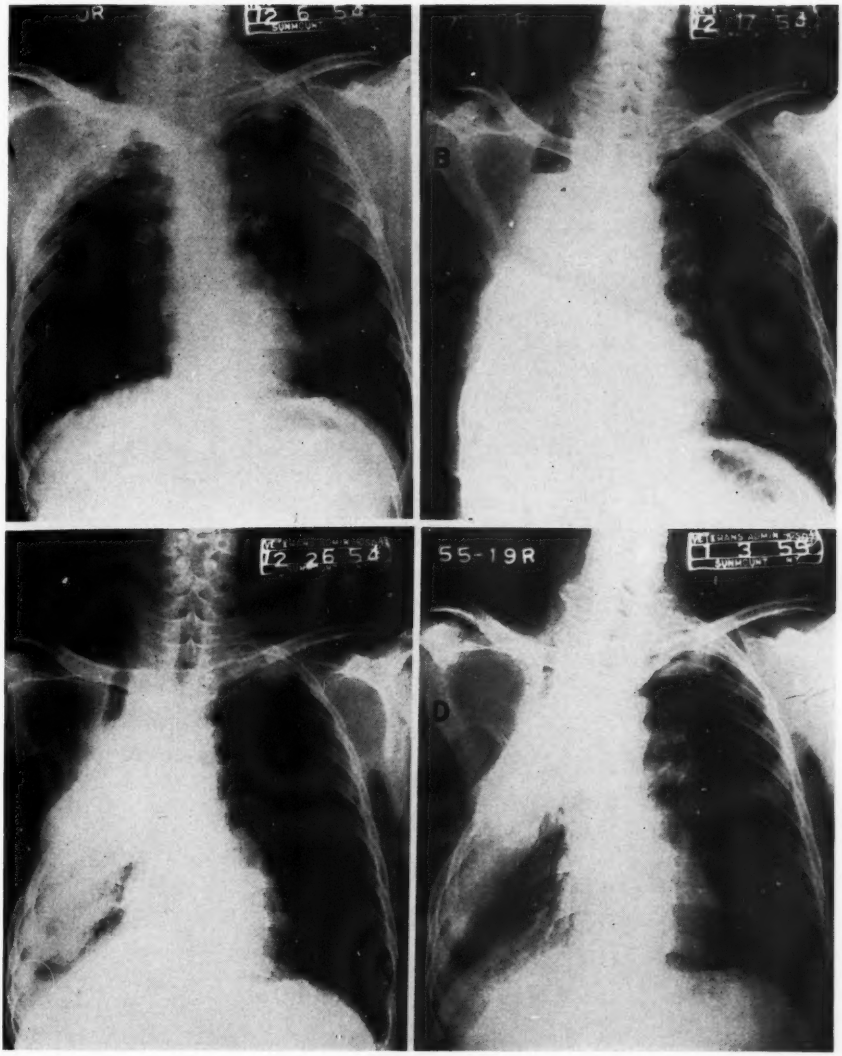

Fig. 1.—*A*, Right preresection thoracoplasty, one week before resection. *B*, four days following right upper lobectomy. *C*, Thirteen days postoperation. *D*, Twenty-one days postoperation.

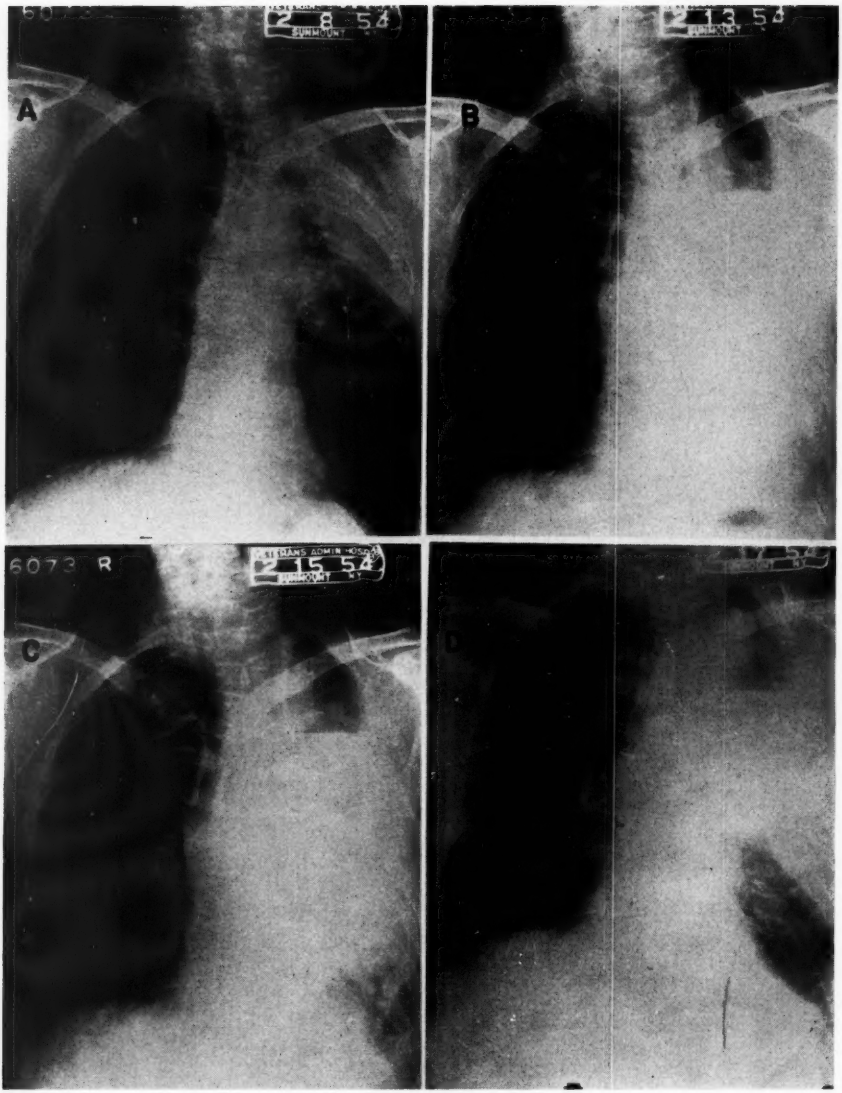

Fig. 2.—*A*, Left preresection thoracoplasty two days before resection. *B*, Three days following upper lobectomy (3-rib thoracoplasty increased to 5 ribs). *C*, Five days postoperation. *D*, One week postoperation. (Bronchospirometry at six months postoperation revealed a trapped left lower lobe, with 10 per cent O_2 uptake.

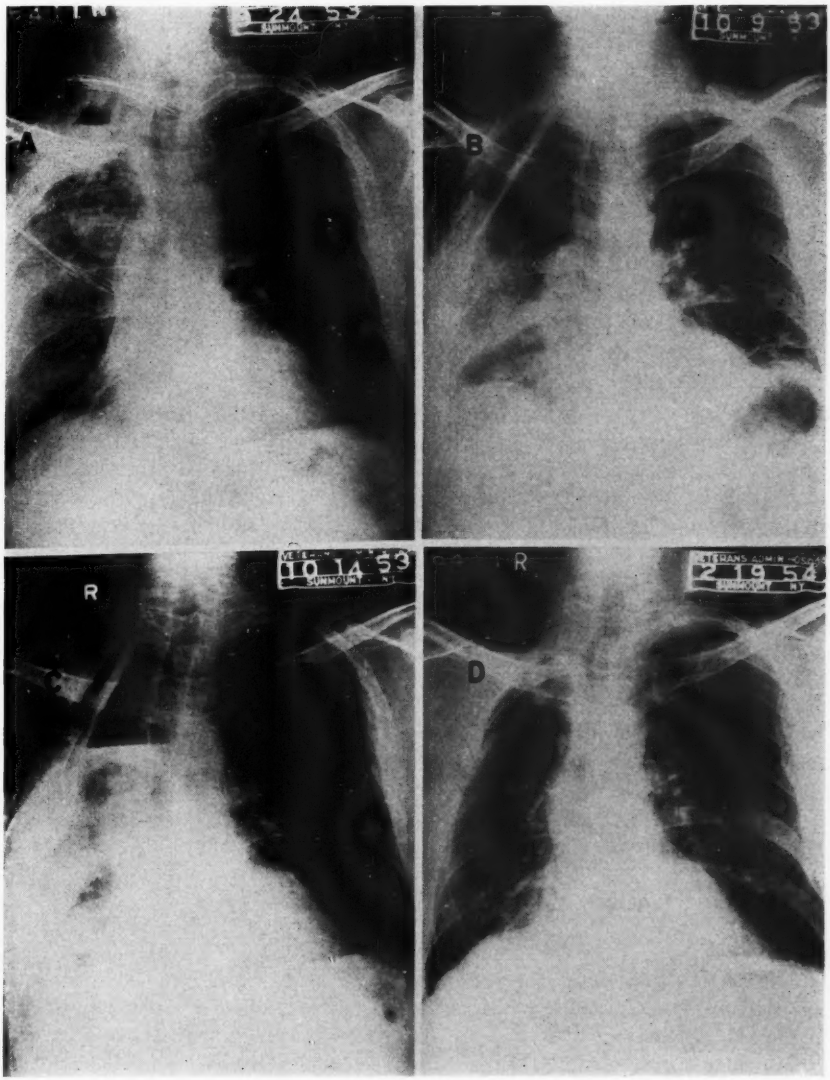

Fig. 3.—*A*, One day following right preresection thoracoplasty. *B*, One day following right upper lobectomy. Note communication with subscapular space. *D*, Six days postoperation. *E*, Four months postoperation. Small residual apical space.

extraperiosteal plombage or osteoplastic procedure of Björk.[8] Since the mechanical disadvantage to the patient is essentially the sum total of both procedures, it is only the young patient with good reserve who can stand this combined insult.

In 11 patients receiving concomitant thoracoplasty following upper lobectomy, Sze, Samadi, and Conant[9] reported postoperative atelectasis in 7. Similarly, J. K. B. Waddington[10] recorded varying degrees of ipsilateral atelectasis in 9 of 20 patients following thoracoplasty concomitantly with resection.

PLEURAL TENTS

Fashioning of a cap or tent of parietal pleura to lower the apex of the chest, and aid in covering raw lung surfaces, has been a valuable adjunct.

The technique of pleural tenting has been described by Miscall and Duffy.[11] It is similar to the creation of an extrapleural pneumothorax, with the exception that it is much easier to perform from within the thorax.

While small rents in the pleura may be sutured, any extensive defect incurred from entry into the extrapleural plane, during mobilization of the upper lobe, makes formation of an intact tent difficult or impossible.

It is possible to tailor the pleural dissection in such a manner that it may be posterolateral, apical, or anterolateral, and overlie the area of resected lung and maximum raw surface. Construction of such a tent adds only 15 minutes to the resection time. The effectiveness of this temporary roof may be estimated before the chest wall is closed.

No effort is made to maintain an extrapleural space with air or liquid during the postoperative period. The space is quickly filled with blood if the pleura remains intact, and shrinks gradually during the successive two or three postoperative months (Fig. 4). In a few cases, however, an apical air pocket (extrapleural) has persisted, and acting on the observation that such a space might indeed be preventing postoperative expansion of the residual lung, we have carried out transient aspiration with needle or catheter.

Our over-all results in this group (25 cases) have been most satisfactory. Whenever it has been used, the pleural tent has been effective in minimizing a potential postresection space and has assisted in reducing the incidence of transient air leakage from the raw surface of lungs with limited pneumatic (self-sealing) function.

POSTRESECTION THORACOPLASTY

A commonly accepted indication for postresection thoracoplasty in tuberculosis surgery has been a persistent apical space unfilled by remaining lung. Although the existence of such a space is usually, but not always, associated with a bronchopleural fistula,[12] the adjunctive "space-filling" thoracoplasty has often been successful in the dual role of space obliteration as well as fistula closure.

Sze, Samadi, and Conant,[9] in a review of their resection group, formerly used a 3- or 4-rib thoracoplasty routinely, following upper lobectomy, ostensibly to prevent overexpansion of the remaining lung and reactivation of residual

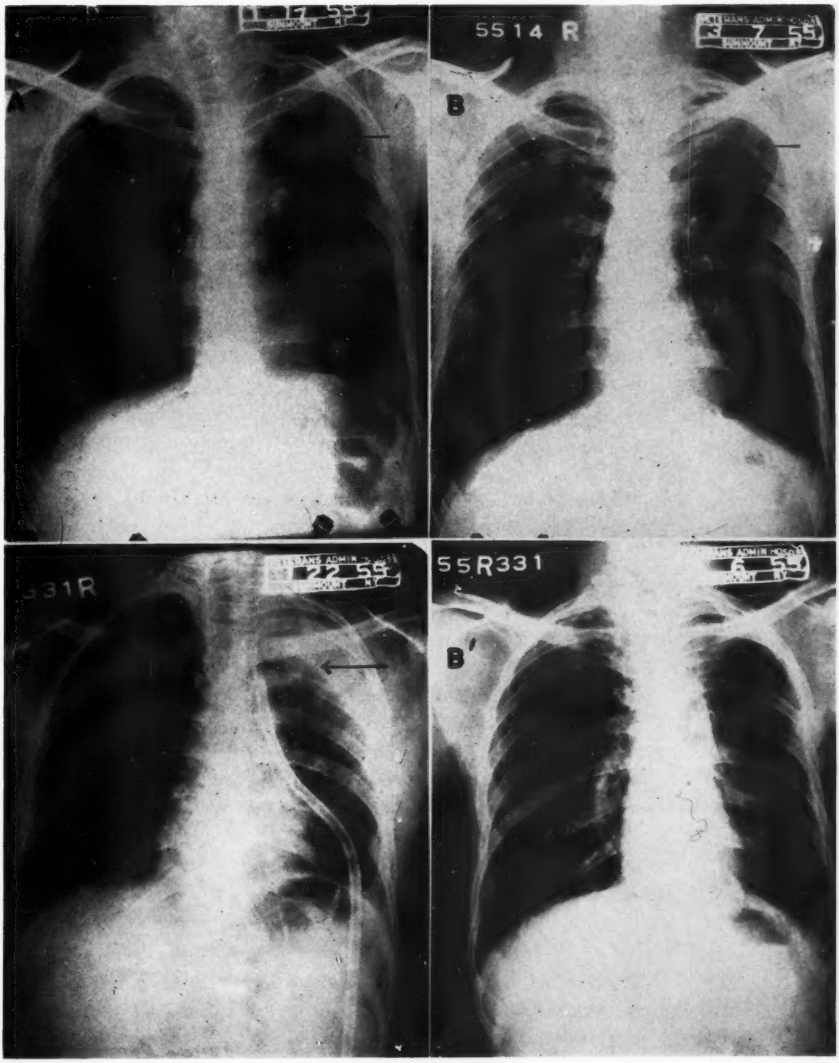

Fig. 4.—Pleural tents. *A*, Twelve days following excision of superior division, left upper lobe. *B*, Two months postoperation. Lung at level of 5th rib posteriorly. *A'* Two days after left upper lobectomy. Tent at level of resected rib. *B'*, Five and one-half months after left upper lobectomy. Pleural tent has disappeared. Left lower lobe filling hemithorax.

disease. At present, with the protection afforded by postoperative chemotherapy for six months or longer, they reserve the postresection thoracoplasty for the occasional patient with residual pleural space or bronchopleural fistula.

Murphy[13] reserved the subsequent thoracoplasty for the case with incipient or frank bronchopleural fistula, or persistent delay in re-expansion. Following this policy, he obtained 82.7 per cent satisfactory results after 98 operations (segmental resections) without a thoracoplasty.

Our indications for the postresection thoracoplasty have been discussed previously[14] and, in general, are similar to those of Murphy.

The need for a small "space obliterating" apical thoracoplasty, subsequent to resection, appears to decrease proportionately as the mechanical problem of dealing with the cut surface of the lung is solved.

The incidence of early or late tuberculous complications in the patient receiving long-term drug therapy, and coming to surgery with susceptible bacilli is quite small. We believe that the indications for a postresection thoracoplasty should now include the bacteriologic consideration of the patient's sputum preoperatively, the presence of resistant bacilli in the sputum or resected material, and the distribution, character, and amount of residual parenchymal disease.

Recently we have been attempting surgical treatment in a group of patients with cavitary upper lobes, positive or intermittently positive sputum, and resistant bacilli. In this group (now numbering 12 patients), we have been aware of the impossibility of removing all palpable disease by preoperative laminography.

The cavity-bearing upper lobe has been removed, leaving residual closed lesions (caseous, necrotic foci) in the lower lobe and contralateral upper lobe, up to, and in some cases, exceeding 2 cm. While the lower lobe may undergo some compensatory emphysema (or already has obstructive emphysema), the contralateral upper lobe may do the same with shifting of the superior mediastinum. In this group with resistant bacilli in the resected specimen, and usually in the regional lymph nodes, we have advised postresection thoracoplasty, largely on the basis of poor chemotherapeutic control rather than the fear of overdistention leading to subsequent reactivation.

DISCUSSION

Björk[15] lists the disadvantages of a preliminary thoracoplasty with secondary resection as follows:

1. Psychological handicap of facing a second major operation.
2. Prolonged hospitalization.
3. The danger of spread is greater after a thoracoplasty than after a resection.
4. It is more difficult to model the chest in the desired size at a second operation.

The physiologic evidence of the handicap which a preresection or concomitant thoracoplasty imposes on a patient is considerable. Björk[16] observed several

instances where 5-rib thoracoplasties with apicolysis resulted in more severe postoperative courses than resection in similar cases. He believed this was due to the combination of compression atelectasis, produced by the thoracoplasty, and the accumulation of fluid in the apicolysis space, plus paradoxical movements of the unstable chest wall. In a series of 16 patients, following resection of segments and lobes under old thoracoplasties, he found that in patients with good pulmonary function, the arterial O_2 tension reached the preoperative value on the fifth day after surgery, whereas in those with advanced bilateral disease, the arterial O_2 tension had not reached the preoperative values after fourteen postoperative days.

W. W. Stead and co-worker,[17, 18] in a series of valuable physiologic studies following thoracic surgery, confirmed the work of Björk and Berggren[32] in finding a fall of hemoglobin saturation by a shunt of blood through recently collapsed lung tissue which was not being aerated. They found, as had other investigators, the existence of a definite respiratory acidosis at the completion of the operative procedure. They determined that in order to correct this acidosis, an increase in alveolar ventilation was required. In the patient who was unable to effect this increase in minute ventilation, because the tidal exchange was limited by pain and varying degrees of paradox, the compensation was met by the more inefficient mechanism of a marked increase in the respiratory rate. In patients who were unable to meet this demand of extra respiratory work because of age, debility, extent of surgery, or associated pathology, which had already compromised their reserve, serious degrees of pulmonary insufficiency or death might result in the postoperative period.

Björk considers the only advantage of staging the thoracoplasty and resection to be the relative absence of paradox at the second procedure. In view of the manifest psychological, surgical, and physiologic disadvantages, there must be more than the lack of paradox to justify the widespread impression that a preliminary thoracoplasty makes resection more safe for a patient.[19] Are there certain patients in whom the drugs are unable to effect the necessary resolution of disease, so that the patient is a better risk for resection? Should a collapse procedure be carried out on these patients with the anticipation of failure therefrom, and the deliberate intent to resect later in any event?

Since a satisfactory thoracoplasty (Alexander or plombage) may arrest up to 80 per cent of cases, it is difficult for us to determine which will be the more unfortunate 20 per cent, without allowing the test of time. If preresection thoracoplasties are done as a routine measure in the open positive group, will not a considerable number of patients have resections whose disease might well have become arrested with the thoracoplasty alone?

In 1952, Takaro, Clagett, Hodgson, and Carr,[20] in reporting 73 cases of pulmonary resection for tuberculosis, with a follow-up of one year or longer, stated that "it is generally agreed now that thoracoplasty should be performed after pneumonectomy or lobectomy for tuberculosis, to prevent overexpansion of the remaining pulmonary tissue and breakdown of quiescent tuberculous

foci.'' This dogma, echoed by many writers in the ensuing years, has become, possibly through strength of repetition, a traditional indication for a concomitant or postresection thoracoplasty.

There are no adequate physiologic or pathologic data to support the contention that true emphysema develops in the remaining lung tissue following lobectomy. Experimental studies,[21, 22] which are largely based on pneumonectomies in dogs, reveal the presence of simple distention, and, later, compensatory emphysema. Whether these changes progress to emphysema in the anatomic sense, with atrophy and rupture of alveolar septa, following *upper lobectomy* in young patients, has not been conclusively demonstrated. Nor is there any good evidence that this amount of lower lobe hyperinflation is a physiologic deficit, or, for that matter, can necessarily be prevented by a postlobectomy thoracoplasty.[22-24] Various degrees of obstructive or localized hypertrophic emphysema already exist in these lungs at the time of resection, apparently as a result of the healing processes of tuberculosis.

If the emphysema-preventing thoracoplasty is to be advocated on a prophylactic basis, it would seem that substantial evidence should be available that such lower lobe distention is a physiologic handicap, and, moreover, that such late changes may be prevented by thoracoplasty.

There have been sporadic case reports of tuberculosis beginning in upper lobes following lower lobectomy for bronchiectasis.[25-27] In almost every reported case, there was no evidence of tuberculosis on the pre- or immediate postoperative chest films. Progressive tuberculosis appeared in the majority of these cases within one year after lobectomy. Rothstein and Gerson[25] attribute this activation of tuberculosis to overexpansion of the upper lobe. However, none of the authors refer to their gross findings at operation, and all cases were done without the benefit of any specific antituberculosis chemotherapy.

We should like to emphasize the extreme unpredictability of the residual necrotic focus. Reactivation of tuberculosis has occurred under a variety of conditions. There is no information in the previous reports which establishes a cause-and-effect relationship of overdistention on small residual lesions in the remaining lobe.

In addition, we have seen 2 patients who had a postlobectomy thoracoplasty and were treated for six months postoperatively, yet had lower lobe reactivation three years later.

In this regard we accept Medlar's[28] concept of pathogenesis; i.e., the spread of tuberculosis is due to bronchogenic dissemination. He has furnished pathologic evidence which reveals that, while the healing process takes place to a certain extent in a collapsed lung, the necrotic lesion is irreversible and maintains its proximity to the bronchus. While actual sloughing of the parenchymal lesions may be prevented by certain mechanical conditions, true healing is rarely seen. He found, in 1 case, a chronic, sloughing, open cavity, with bronchial communication, which had been under a thoracoplasty for fifteen years. Dr. Medlar[29] states further, he has never seen a specimen in which reactivation from a latent caseous lesion was conclusively due to the process of overexpansion.

In conclusion, it would seem that if adjunctive thoracoplasty is to prove its value in association with resection surgery for tuberculosis, there must be definite evidence of protection against major tuberculous complications in the thoracoplasty group. Complications (fistula, empyema, spread) following lobectomy (411 cases), with and without previous thoracoplasty, as reported in one year of tabulated resection data from 47 contributing hospitals (14th VA, Army-Navy Conference on Chemotherapy of Tuberculosis)[30] do not indicate that any protection is afforded by the preresection thoracoplasty. Overholt, Wilson, and Gehring[31] concluded in 1952, that, "Chemotherapy has produced a profound reduction in the risk associated with these operations. Thoracoplasty done before, with, or immediately after resection, did not lower the complication rate in our series." If this statement was true in 1952, it would appear even more justified in 1955.

SUMMARY

1. A series of 29 cases of preresection thoracoplasty, 25 cases of pleural tent, and 25 of postresection thoracoplasty is presented with their clinical indications.

2. The physiologic handicap of the preresection and concomitant thoracoplasty is emphasized.

3. There is no definite pathologic or clinical evidence that the adjunctive thoracoplasty makes resection more safe for a patient.

4. The impression that reduction in the size of the hemithorax should benefit patients with obstructive or compensatory emphysema is not tenable. These are the patients who are most vulnerable to any interference with the bellows mechanism of thorax and diaphragm.[7]

5. Prophylactic thoracoplasty, for the sake of preventing overdistention or reactivation of tuberculosis is a nebulous concept. A thoracoplasty for the purpose of closing a fistula or empyema space is therapeutic.

6. The foregoing statements are not presented with any intent to *settle an issue*. The plea is made that, if a patient is to be given an adjunctive thoracoplasty, there must be considerably better testimony than is now available that it is worth the price he pays.

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THE PERSISTENT PLEURAL SPACE FOLLOWING PARTIAL PULMONARY RESECTION

ELLSWORTH E. WAREHAM, M.D.,* HUGH BARBER, M.D., JAMES S. MCGOEY, M.D., AND LAURENCE MISCALL, M.D.
NEW YORK, N. Y.

INTRODUCTION

ALTHOUGH the goal in the postoperative management of patients having partial pulmonary resection is to re-expand the lung rapidly and bring it into apposition with the chest wall, there are a certain number of patients where this is not accomplished. A persistent pleural air space is not infrequently an x-ray finding after partial pulmonary resection. This finding is a cause of concern for the surgeon. The hazard of infection, especially suppuration with attendant extension into other parts of the lung or pleural space, prompts the surgeon to obliterate the space by early vigorous measures. These include needling and aspiration, closed drainage with or without suction, and, very often, thoracoplasty.

Now, however, with the use of antibiotics many complicating infections may be prevented. This study is a review of data on patients who showed a persistent pleural air space in order to observe the fate of the space with and without secondary surgery.

PATHOGENESIS

An early postoperative space may be due to a disproportion in volume between the chest cavity and the remaining lung tissue. Without an air source, this type of space gradually contracts, fills in with exudate, and obliterates. A pleural space which persists is strong evidence of bronchial air leakage. If the air source is large as from a lobar or segmental bronchus, this fistula results in an infected space. However, if the air source is small as from a terminal or respiratory bronchiole, and the patient is under antibiotic treatment, there is a high probability that the patient will be symptomless and the space would go unrecognized but for the finding on chest roentgenogram.

METHOD

All patients undergoing partial pulmonary resection at Triboro Hospital between July 1, 1951, and June 30, 1953, were studied. Selected preoperative x-rays were scanned for the type of disease, and all postoperative x-rays of each patient were serially reviewed to determine whether there was a pleural

From the Department of Thoracic Surgery, Triboro Hospital, Jamaica, N. Y.
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*Present address: 1720 Brooklyn Avenue, Los Angeles 33, Calif.

space. When a space was present twenty-one or more days after operation, it was recorded as a persistent space. Subsequent films were examined to determine the fate of the space and how long it persisted. At the same time the clinical record was investigated in detail.

These patients were nontuberculous and tuberculous. In the tuberculous patients, if organisms were found in the sputum or gastric contents within ninety days before the day of surgery, the patient was classified as sputum positive. The remaining are tabulated sputum negative.

Preoperatively, all tuberculous patients received combined long-term chemotherapy until maximum benefit was obtained, determined by chest roentgenogram and sputum studies. Postoperatively, drug therapy was continued for at least four months. The nontuberculous patients received suitable chemotherapy before and after surgery. In all cases, the decision for surgery was made at a medical-surgical staff conference.

Postoperatively, the patient's course was noted for fever, tachycardia, respiratory symptoms—especially cough, hemoptysis, wheeze, sputum both as to volume and character—and body weight. The persistence and amount of air leakage through the chest drainage tubes as well as the character of the fluid drainage were observed. Supplementary surgical procedures were tabulated, and in tuberculous patients the sputum was examined and cultured for bacilli. These observations extended from the time of surgery to the last follow-up visit. This represents a follow-up from one and one-half to three and one-half years.

PRESENTATION OF DATA

This study comprises 136 patients (Table I) who had partial pulmonary resection. There were 104 procedures for pulmonary tuberculosis, of which 33 cases were sputum positive. There were 32 operations for nontuberculous pulmonary disease.

TABLE I. TYPES OF PARTIAL PULMONARY RESECTION IN 136 PATIENTS

		TUBERCULOUS		NON-TUBERCULOUS	COMBINED TOTAL
		SPUTUM			
		POSITIVE	NEGATIVE		
Lobectomy		14	25	20	59
Segmental		12	33	5	50
Wedge		0	1	4	5
Lobectomy and	Segment or Wedge	7	12	3	22
Total		33	71	32	136

After study of all postoperative roentgen chest films on these patients, which included tomograms in the anterior-posterior and lateral projections, 41 were found to have a pleural space twenty-one or more days after lung resection. Without the routine use of sectional radiography, the identification of many of these pleural spaces would have been difficult, particularly so in determining a space under a thoracoplasty. Postoperative fibrotic strands and

emphysematous lung and blebs have been hard to distinguish from a space without this aid. A considerable number of these spaces would not have been detected on conventional posterior-anterior and lateral films.

The pleural air spaces were evident generally from the first day following lung resection and could be followed throughout their course. Several, however, appeared a week or more after surgery, and some after removal of the chest drainage tubes.

Postoperative spaces were followed on serial roentgenograms and the time of their disappearance recorded. Such patients have been followed postoperatively for a period of from one and one-half to three and one-half years. All are still being followed except for two who did not return to the clinic after disappearance of the space on x-ray.

Clinical records of the 41 patients with pleural spaces were examined in detail. After operation, 14 of these had persistent symptoms such as cough, wheeze, sputum production, fever, tachycardia, and weight loss. These patients have been classified arbitrarily as having bronchopleural fistulas. The remaining 27 asymptomatic patients make up the group of particular interest in this study. They had an uneventful postoperative recovery and on clinical grounds could be considered uncomplicated. The presence of a space on x-ray was not anticipated.

ASYMPTOMATIC GROUP—PERSISTENT PLEURAL SPACE

Table II classifies these asymptomatic patients according to type of resection and Fig. 1 shows the type of disease which they had at the time of pulmonary resection. There were 20 operations for pulmonary tuberculosis. Six of these patients had positive sputum. The remaining seven operations were

TABLE II. INCIDENCE OF ASYMPTOMATIC PLEURAL AIR SPACES IN 136 PATIENTS ACCORDING TO TYPE OF PULMONARY RESECTION

	TUBERCULOUS		NON-TUBERCULOUS	TOTAL	PERCENTAGE WITH SPACES (PER CENT)
	SPUTUM				
	POSITIVE	NEGATIVE			
Lobectomy	0	4	2	6	10.2
Segmental	3	6	2	11	22.0
Wedge	0	0	3	3	60.0
Lobectomy and Segment or Wedge	3	4	0	7	33.2

for nontuberculous pulmonary disease. It will be seen that the incidence of spaces in segmental and wedge resections, or combinations of these, exceeds that from lobectomy. This is because the parenchymal dissection inherent in the procedure involves a greater surgical surface of lung tissue from which air leakage occurs.

Fig. 2 shows how long the spaces persisted on tomographic roentgenogram. One-half of them had disappeared before five months. There were seven cases which were followed for a period of over seven months (Fig. 3). Three had spaces which were persistent after two years. All these patients

are on full activity and remain free of symptoms. One tuberculous patient was found on follow-up, ten months after discharge, to have a cavity in the contralateral lung. The pleural space had been closed for six months, however, and the ipsolateral lung parenchyma remained free of disease. The sputum had also been negative during this time.

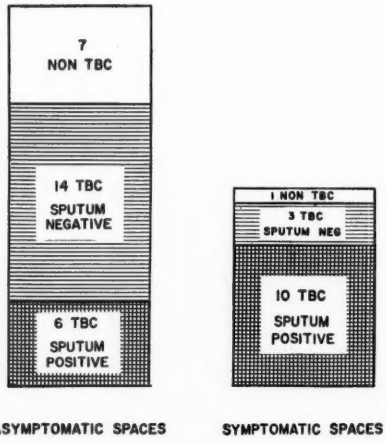

Fig. 1.—Type of disease in patients having spaces.

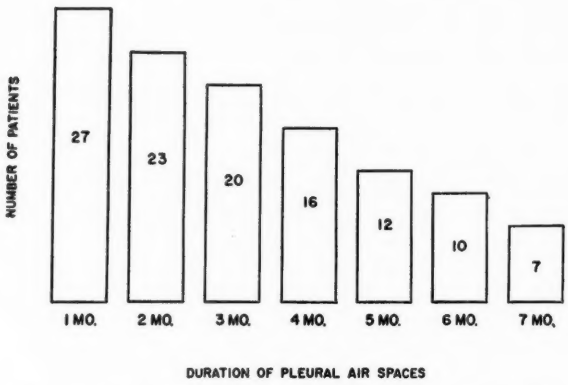

Fig. 2.—Duration of the pleural air space in 27 asymptomatic patients. Three patients have shown a persistent pleural space for over two years.

In reviewing the surgical management of this group without symptoms, it was found that 20 patients received no supplementary surgery although at conference the advisability of thoracoplasty was considered regularly. One patient had a limited thoracoplasty at the time of resection. Several patients refused further surgery because of their well-being. There were seven who had operative intervention. One had catheter drainage for twenty-four days.

Six had surgical exploration for the pleural space and limited rib resection over the space. Three of the patients in whom thoracoplasty was carried out continued to have the space for one year or more after the secondary procedure.

Exploration of the spaces in the 6 asymptomatic patients, just mentioned, revealed findings which were quite uniform. Their size varied from 3 to 10 cm. They were located in the upper half of the chest between the operated surface of the lung and the chest wall. During the first four to six weeks, the space was covered with exudate and organizing tissue. Minute openings could be demonstrated on the surgical lung surface from which air leaked with positive endotracheal pressure. Those spaces opened six months or more after resection were found to be endothelized and had a cystlike appearance (Fig. 4). The same minute bronchiolar openings could be demonstrated. None of these asymptomatic spaces explored was found to contain fluid or evidence of infection. Cultures for pyogenic organisms and tubercle bacilli did not show growth.

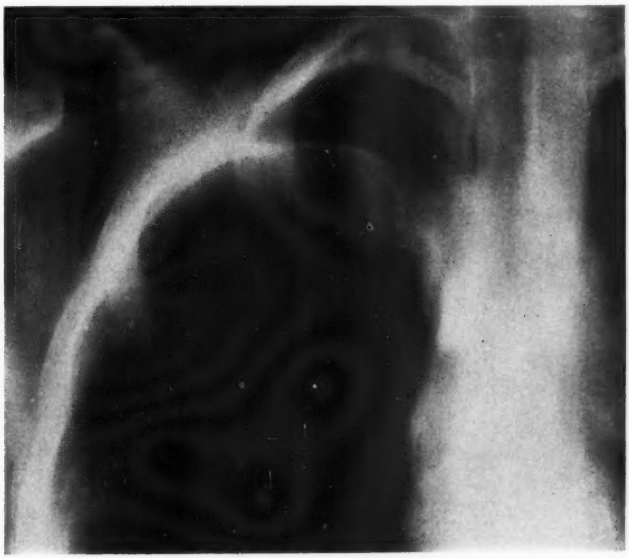

Fig. 3.—Tomogram of asymptomatic apical pleural space three months after resection of apical and posterior segments for bullous cysts. After ten months of observation it disappeared to x-ray view.

SYMPTOMATIC GROUP—BRONCHOPLEURAL FISTULA

Table III shows the distribution according to the type of resection of 14 patients with symptoms of bronchopleural fistula and Fig. 1 shows the type of disease they had at the time of pulmonary resection. There were 13 operations for pulmonary tuberculosis, ten of which were in patients who had tubercle bacilli in the sputum. The one nontuberculous patient had a destroyed

Fig. 4.—Tomogram of asymptomatic apical pleural space which was explored eleven months after resection of the left apical posterior segment for cavitary tuberculous lesion. The space was found epithelized and cystlike in appearance.

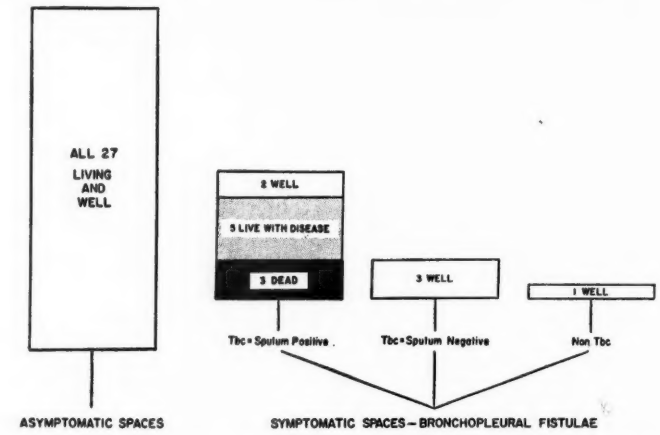

Fig. 5.—Present status of patients with asymptomatic and symptomatic spaces followed for one and one-half to three and one-half years.

TABLE III. INCIDENCE OF SYMPTOMATIC PLEURAL SPACES (BRONCHOPLEURAL FISTULAS) IN 136 PATIENTS ACCORDING TO TYPE OF PULMONARY RESECTION

	TUBERCULOUS SPUTUM		NON-TUBERCULOUS	TOTAL	PERCENTAGE WITH FISTULAS (PER CENT)
	POSITIVE	NEGATIVE			
Lobectomy	5	0	1	6	10.2
Segmental	4	2	0	6	12.0
Wedge	0	0	0	0	0.0
Lobectomy and Segment or Wedge	1	1	0	2	9.0

middle lobe. There is in our group an approximate 10 per cent incidence of bronchopleural fistula, the incidence being similar for segmental resection and lobectomy. All these patients have had prompt chest drainage, multiple stage thoracoplasty, and various plastic procedures. Some have had further pulmonary resection and others, early secondary closure of an open bronchus.

Despite aggressive surgical attack, it will be noted in Fig. 5 that after a follow-up from one and one-half to three and one-half years on the ten patients with positive sputum that three are dead, four having persistent fistula live with disease in the hospital, and 1 lives at home. In the remaining two, the fistulas have closed and the patients appear well.

The three tuberculous patients with negative sputum who had bronchopleural fistulas are free of symptoms and fully active. Two of these continue to have apical spaces under a thoracoplasty. The remaining one, two months after upper lobectomy, had a secondary closure of an open bronchus with complementary thoracoplasty which obliterated the space. She also is well.

One nontuberculous patient with a bronchopleural fistula healed after open drainage and thoracoplasty.

DISCUSSION

We are impressed with the uneventful recovery of those asymptomatic patients with negative sputum who show on x-ray a persistent pleural space after partial pulmonary resection. None of these patients to the present time has developed complications because of the space. Prior to the antibiotic age, infection would be anticipated and hence obliteration of the space by operative intervention strongly advocated. The need for this additional surgery as a routine may now require evaluation, for the majority of these spaces will obliterate in a few months, and there appears to be little risk in following them for a year or more. A decrease in size of the space on serial roentgenograms is a favorable sign.

On the other hand, a patient with a pleural space whose sputum continues positive for organisms, in spite of drug therapy, has an uncertain prognosis for recovery. All deaths and serious morbidity in our series occurred among patients infected with antimicrobial resistant organisms (Fig. 5).

SUMMARY

The postoperative x-rays of 136 consecutive patients undergoing partial pulmonary resection were scrutinized for a pleural air space present twenty-one or more days after operation.

There were 41 patients in whom a persistent pleural air space was found and the significant clinical data were correlated.

Soon after operation, 14 of these patients had respiratory and constitutional symptoms indicative of bronchopleural fistula, ten of whom were infected with drug resistant tubercle bacilli. For the latter group, the prognosis was poor despite multiple operations; three are dead, five live with disease, and only two are clinically well. The remaining four patients whose infective organisms were preoperatively controlled by chemotherapy are clinically well after supplementary surgery.

There were 27 patients with pleural air spaces who were asymptomatic. They have had a satisfactory recovery with or without supplementary surgery.

This study shows that a postoperative pleural air space may be safely treated conservatively if the patient is carefully observed and the following conditions obtain:

1. There must be absence of respiratory and constitutional symptoms.
2. There should be progressive shrinkage of the space on serial roentgenograms. Tomograms are very helpful in this follow-up.
3. In tuberculous patients, the sputum must be negative for tubercle bacilli.
4. The patient should be on effective antibiotic drug therapy for control of the parenchymal disease.

If these criteria are not met, the patient should have early surgical intervention to obliterate the space.

THE MANAGEMENT OF THE PLEURAL SPACE FOLLOWING PARTIAL RESECTION OF THE LUNG (ESPECIALLY FOR TUBERCULOSIS)

CHARLES W. FINDLAY, JR.,* M.D.
NEW YORK, N. Y.

INTRODUCTION

THE obliteration of the pleural cavity following partial removal of a lung is most important. Alert surgeons strive for this ideal as early as possible in the postoperative period. A pleural space that has persisted for more than two weeks may lead to chronic empyema with or without bronchopleural fistula. Such complications often require further deforming and sometimes difficult operations, and the convalescence of the patient is prolonged. With eventual healing, fibrosis of pleura and diaphragmatic deformity may lessen future pulmonary function of the operated lung.

At Fitzsimons Army Hospital, over four hundred lobectomies or partial lobectomies were done during the past two years. Two-thirds of these involved the resection of lobar segments that was performed usually for tuberculosis. It is very important that the postoperative tuberculous patient recovers without a pleural space or bronchopleural fistula. Such residuals usually prevent ultimate cures of the disease and lead to difficult problems in total management. It has been noted, however, that this group has recalcitrant pleural difficulties which tax the ingenuity of the surgeon. The Thoracic Surgical Service at Fitzsimons Army Hospital for the past two years has included students of this specialty from different parts of the country. They have contributed to the evolution of management of the pathology in question. Considerations for the care and prevention of pleural spaces after subtotal pulmonary rsection follow.

THE ADJUVANTS AND CAREFULNESS OF SURGERY AT THE TIME OF RESECTION

The type of operation and the amount of lung to be removed should be planned as completely as possible before surgery. Ideally, the lung remaining after resection should be free of inflammatory and mechanical disease. A bronchiectatic remnant does not expand well postoperatively and it is prone to develop areas of pneumonitis. The suturing of a divided bronchus with a tuberculous ulcer may result in a fistula later. These factors and many others must be diagnosed beforehand so that proper surgery may be selected.

From the Surgical Service, Thoracic Surgery Section, Fitzsimons Army Hospital, Denver, Colo.

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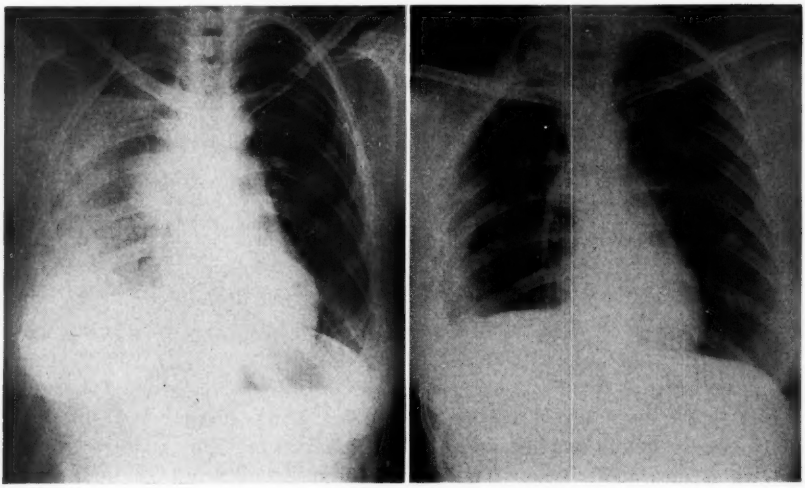
*Instructor in Surgery, Columbia University; formerly Captain, MC, A.U.S.

Preliminary thoracoplasty in tuberculous patients is a subject of much debate. Some surgeons prefer to use it at the time of resection or as a later procedure. At Fitzsimons, a modified thoracoplasty is done about three weeks prior to resection if the removal of more than two segments of an upper lobe is contemplated. The posterior half of the third rib and all of the second rib are resected subperiosteally leaving the transverse processes intact. The periosteum is stripped from the undersurface of the first rib and an apicolysis is performed down to the level of the third or fourth rib posteriorly (Fig. 1, *A* and B). If three or more segments are excised without this preliminary step, the modified thoracoplasty is done several weeks later. Concomitant procedures are not done unless the equivalent of four or more segments is removed and a first-stage thoracoplasty already has been done. Under such

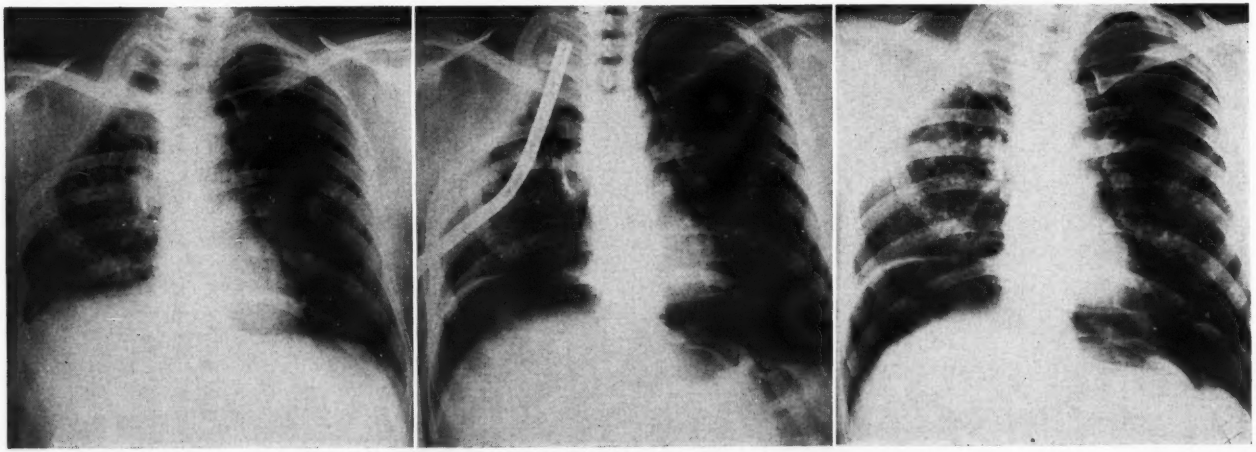


A. *B.*

Fig. 1.—*A* (Sept. 10, 1954). On Aug. 16, 1954, the patient had a modified right upper thoracoplasty. On Sept. 8, 1954, she had right upper lobe lobectomy for tuberculosis. On Sept. 10, 1954, the chest film showed a significant air-fluid collection in the right chest and the tubes were not draining well.

B (Dec. 6, 1954). SK-SD solution was instilled into the chest through the upper and lower tubes. The fluid and air were aspirated three hours later. This was done three times during the first postoperative week. Although the treatment caused considerable pleural reaction, the pleural space was effectively evacuated. Most of the pleural thickening had disappeared by Dec. 6, 1954.

conditions a second stage may be completed at the time of resection. An example would be the excision of an upper lobe and the superior segment of the lower lobe in a patient that had a preliminary thoracoplasty. The fifth rib would be removed for the intrathoracic procedure and a posterior segment of the fourth rib would be removed near the end of the operation (Fig. 2, *A*). There is minimal paradoxical motion following this method of management. The relaxed chest wall approaches the small lung remnant and often leads to early obliteration of the pleural cavity. It also prevents overexpansion of the underlying lung.



A. *B.* *C.*

Fig. 2.—*A* (Nov. 8, 1954). On Oct. 4, 1954, a modified right upper thoracoplasty was performed. The patient had a right upper lobe lobectomy and resection of the superior segment of the right lower lobe on Oct. 26, 1954, for tuberculosis. A five-rib thoracoplasty was completed at the time of surgery. Postoperatively, he had a persistent apical space despite repeated needle aspirations and the use of indwelling polyethylene catheters. A small fistula was present.

B (Nov. 12, 1954). On Nov. 10, 1954, a tube was inserted to the tip of the apical space through the anterolateral chest wall under direct vision. The subscapular space was avoided, and pneumoperitoneum was started. The pleural space was obliterated soon and the fistula closed thereafter.

C (Nov. 30, 1954). By Nov. 30, 1954, he was convalescing well and there was no recurrence of the space.

Attention to details at the time of resection contributes to early evacuation of the pleural space and cessation of air leaks. The lysis of pleural adhesion, especially in the lower half of the chest, permits a free egress of fluid and air through the drainage tubes. It also allows for uniform expansion of this lung remnant. Sometimes, however, it is possible and judicious to leave some of the apical adhesions intact to prevent later space problems in that area.

If segmental surgery is performed, the remaining raw segmental surface should be free of disease. The blunt dissection used during the removal of a segment is difficult if inflammation or fibrosis has crossed the intersegmental plane. Deviations occur that either remove too much or too little parenchyma. Devitalized tissue may be left behind or injuries may be made to the bronchi, veins, or lung tissue of the adjacent and remaining segment. Such technical errors lead to localized pulmonary infarction, expansion difficulties, and troublesome air leaks. It is sometimes wise to remove more lung than planned to prevent the above complications.[2]

After the removal of the specimen, any depleuralized lung surface should be examined carefully for bleeding and large air leaks. These should be properly sutured. Small alveolar leaks usually close over soon. There should be minimal distortion of the lung remnant. If there has not been too much suturing of the raw segmental surface and there are no limiting adhesions, a normal lung remnant should expand evenly and early following surgery. All transected major bronchi should be covered with pleural flaps or adjacent tissue. Complete intrapleural hemostasis will be rewarding since it usually obviates postoperative transfusions, frequent chest taps, and later surgery. Near the end of the resective surgery, all segments of the remaining lung should expand well with positive intratracheal insufflation. Any expansile lag of all or part of the lung may be caused by remaining disease, endobronchial material, or operative kinking of a bronchus. Such a finding is best corrected at the time of surgery.

Proper drainage of the pleural cavity is very important. It is wise to use two No. 40 French tubes. The tip of one should be placed as high in the chest as possible to remove air, and the other should be in the most dependent position to remove fluid. They should be so inserted that limitation of pulmonary expansion does not occur. The external ends of the tubes are attached to water-seal drainage to allow good escape of pleural contents during the closing of the wound and thereafter. To prevent early intrapleural clotting, about 300 c.c. of saline are poured into the pleural space just prior to closure. Antibiotics are added to this fluid to combat early postoperative infection.

Concomitant pneumoperitoneum is a valuable adjunct in resection of all or part of the lower lobe. About 700 c.c. of air is instilled into the abdomen after closure of the chest. More air is added later depending on the condition of the patient and the roentgenographic findings. Preservation of the phrenic nerve prevents troublesome paradoxical motion and difficulty with bronchial secretions. More recently concomitant pneumoperitoneum following upper lobe surgery has been used. The rationale for this is upward dis-

placement of the remaining lung which then expands more readily into the apical space. The first few patients treated in this manner have had no space difficulties, but further studies are necessary for complete evaluation.

ACTIVE EARLY POSTOPERATIVE CARE

Aggressive management shortly after surgery is important to prevent pleural space problems. The tracheobronchial airway should be clear at all times. Usually coughing, deep breathing, and frequent changes of the patient's position suffice. Frequently, however, intratracheal suction by catheter is necessary and occasionally blood, mucus, and exudate must be removed by bronchoscopy. A helpful adjunct is the use of the intermittent positive pressure breathing apparatus, which is good for clearing the bronchiolar passages.[3, 5] It may be used in the presence of a bronchopleural fistula provided a pleural drainage tube is in place leading to a water-seal bottle or suction.

Prophylactic treatment against pleural infection is important. Appropriate oral and parenteral antibiotics should be started before surgery and maintained for at least a week after operation. This treatment will also combat pulmonary infection provided atelectasis does not develop. Early apposition of the visceral and parietal pleurae is one of the best preventives against empyema. An obliterated pleural cavity rarely develops infection whereas a persistent pleural space is prone to become infected later.

One of the most pressing problems is the maintenance of adequate pleural drainage. Large rubber tubes are the best conduits to remove intrapleural air and fluid. They should lead at least to water-seal bottles at steady downward gradients. There are advocates of various degress of suction ranging from 6 to 40 cm. of water. Usually 6 to 10 cm. of water suction is adequate, but higher degrees may be used later in the recovery period. Low suction does not cause excessive pleural edema or reaction. When all air and fluid have been removed, the air leaks probably close by their apposition to adjacent surfaces and fibrinous occlusion.

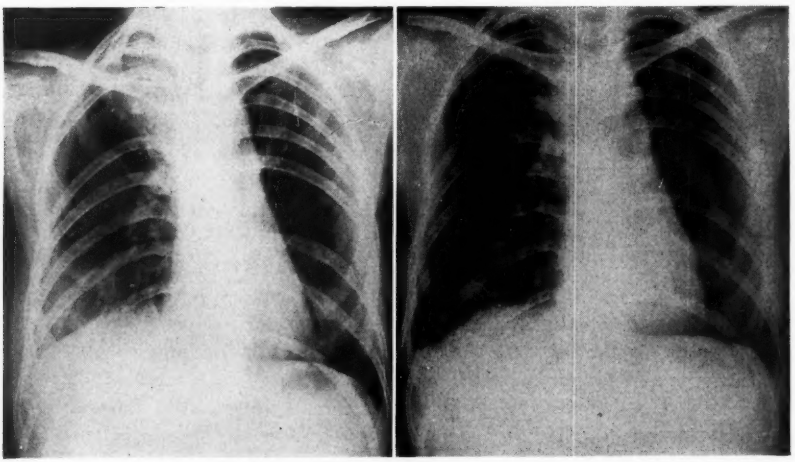
Chest tubes are removed when their fluid columns stop fluctuating and the space is obliterated. This usually occurs from the second to the seventh postoperative day. If a tube is plugged prematurely, it should be milked close to the chest to resume patency. Sometimes, antibiotic solutions or streptokinase-streptodornase (SK-SD) must be instilled into the tube proximally to dislodge or dissolve clots. If a bronchopleural fistula persists, at least one tube usually stays open and it must be kept open until the desired result is obtained. When expanding lung separates the internal opening of a tube from a persistent pleural space that does not have a negative pressure, the tube should be removed and another inserted to the proper place.

THE PERSISTENT POSTOPERATIVE SPACE AT THE PLEURAL APEX

Although a patient has been treated by the above standards, an apical space may persist. This space may be benign, but it may herald future difficulties. It should be considered dangerous until more is known about it. The alert surgeon should evaluate and treat it as soon as possible.

An obstructed tube, especially to the upper chest, in the presence of an apical space is either plugged with fibrin or the internal opening is closed by the expanding lung remnant. When this occurs, the surgeon should know the pressure within the space. This may be done by tapping with a needle the air pocket posteriorly at the proper level. Any fluid is withdrawn if possible. Air is removed until a high negative pressure develops or until it is obvious that a fistula is present.

When a high negative pressure is maintained, the upper tube can be removed. The lower tube is taken out when it cannot be made to function properly or when the lower chest contains no air or fluid. The apical space is studied by serial roentgenograms. When it progressively diminishes in size without fluid collection, nothing further is done. Films will show a convex upper lung remnant rising toward the dome of the chest. If obliteration is slow or fluid recurs, the space may have to be tapped several more times (Fig. 3, *A* and *B*). Antibiotic solutions should be instilled when infection is feared.



A. *B.*

Fig. 3.—*A* (Sept. 17, 1954). In March, 1954, a segmental resection was performed on the left lung for tuberculosis, and the patient made a good recovery. On Sept. 10, 1954, she had an apicoposterior segmental resection of the right upper lobe for the same disease. It was also necessary to remove a wedge of tissue from the adjacent anterior segment. On the fifth day after surgery an apical space was demonstrated by chest films. For the next five days it was aspirated by needle through the third interspace, posteriorly. No fistula was demonstrated and the space closed slowly but progressively.

B (Nov. 23, 1954). Seventy-four days after surgery her film was as shown.

More complicated treatment is necessary when the initial chest tap shows the presence of a fistula by free flow of air through the needle. The same is true when, at the end of tapping, a negative pressure is reached which becomes neutral or positive the next day, indicating a slow bronchial leak. If both tubes are in place, the surgeon may elect to insert streptokinase-streptodornase through the upper tube (Fig. 1).[1, 4] This should dissolve any intraluminal and adjacent clot so that the upper tube again communicates with the apical space.

Following instillation, both tubes are clamped for three or four hours. Relatively high suction is exerted on them when they are opened. The intrapleural fluid and air is drawn off as fast as possible with the hope that the apical space will obliterate immediately. If this ideal is not obtained, constant suction of about 10 cm. of water is maintained until the tubes cease to function.

Occasionally polyethylene tubing has been used to decompress an apical space that is not intubated effectively. Such treatment is used when the fistula is very small. The tubing is inserted posteriorly through a No. 15 gauge needle into the space and attached to low suction. The space has disappeared with this method in a few cases. Usually this treatment has been unsatisfactory due to plugging of the tube despite frequent cleaning with antibiotic solutions. Apparently the egress of air or fluid from the space often is not fast enough to allow expansion of the lung remnant.

At times, an intubated apical space fails to obliterate despite the above management. The functionless upper tube is then removed and another tube is reinserted immediately. This is preferably done through the third interspace in the anterior axilla. The pectoralis muscle is not injured since its lateral border is retracted forward. A closed procedure using a trocar usually suffices. If lung is adherent to the parietal pleura in this region, the intercostal space is opened for a short distance, the pleural symphysis is parted and the tube is advanced into the pleural apex under direct vision (Fig. 2). The wound is closed tightly in layers about the tube.

Sometimes an apical air space under neutral or positive pressure will lead to retraction of the lower lung as well. The roentgenogram will show two spaces with apparent pleural apposition in the mid-lung field. Almost invariably, however, these two spaces communicate. This situation requires both apical re-intubation as described previously and closed intubation of the lower space to ensure fast re-expansion of the lung.

It is probably beneficial to start pneumoperitoneum whenever a space becomes difficult to obliterate if it has not been given already. The adjunct is of value only when there is a negative pressure in the pleural space. This often implies the presence of a good functioning intrapleural tube of adequate size connected to low suction, exteriorly.

In some cases, a drainage tube may remain patent for many days without special care. This usually means that there is a persistent space and relatively large bronchial leaks. If the lung remnant is large enough and expansible, pleural obliteration should appear before long. When signs of pleural infection appear, such as purulent drainage or systemic reaction, an adequate thoracoplasty should be contemplated with the drainage tube in situ.

THE PROBLEM OF THE BASAL SPACE

Following resection of all or part of a lower lobe, there is almost always a space in the lower chest during the immediate postoperative period. Because of its dependent position, the problem of fluid evacuation is important. Any fluid should be withdrawn as early as possible before clotting develops. Other aspects of blood in the lower chest will be discussed later.

The use of concomitant pneumoperitoneum at the time of lower lobe surgery has been most beneficial, and it is used whenever possible. The elevation of the diaphragm diminishes the size of a basal air space, and less expansion of the lung remnant is required to close it. It is re-emphasized that the space is best obliterated by proper intubation with suction.

Rarely, the upper lung remnant is too small or it has unrecognized disease so that it will not expand into a basal space. If good drainage and pneumoperitoneum are of no avail, an upper thoracoplasty or plombage may be performed. The lung is shifted downward by these operations and less expansion is needed to fill the basal space.

A pleural space problem in the lower chest worthy of special mention is that which appears next to the mediastinum. Such spaces usually contain both air and fluid, and the fluid level seen by roentgenography may not extend to the chest wall anywhere. Paramediastinal air and fluid collections usually develop originally by the dissection of air from a bronchial leak or an undrained air pocket elsewhere in the same pleural cavity. If such a space is inadequately treated, it will soon loculate. Tapping the area may injure adjacent lung and the placement of a drainage tube would have to be in an awkward location.

It is recommended, therefore, that an open thoracotomy be done for a paramediastinal collection that does not respond to existing drainage (Fig. 4). This surgery is done preferably before the lung is firmly adherent to the diaphragm or before ten postoperative days have elapsed. A small incision is made in the chest posterolaterally at the level of the diaphragm. If possible, the pleura is entered through an intercostal space, but it may be necessary to remove a short segment of rib. The lower surface of the lung is separated from the diaphragm until the paramediastinal space is entered. A large soft tube is inserted then with the aid of a trocar through the chest wall near by so that its tip rests at the base of the space near the mediastinum. All fluid is removed under direct vision. The original thoracotomy wound is closed and the tube is attached to low suction.

To prevent this complication from reappearing, the surgeon should decompress any inadequately treated air space or bronchial leak elsewhere in the pleural cavity. This may require the insertion of another drainage tube to the proper site and again low suction should be used (Fig. 4, *B* and *C*).

HEMOTHORAX IN THE EARLY POSTOPERATIVE PERIOD

A certain amount of bloody fluid always appears in the pleural cavity following pulmonary resection, and most of it collects during the first twenty-four hours. This creates an abnormal pleural space and the contents should be evacuated before clotting occurs. The installation of 300 c.c. of saline prior to closure delays early clot formation. Dependent constant drainage from the time of surgery usually gives good results.

When there is persistent intrapleural fluid and little or no drainage from the chest tube, thoracentesis should be done as often as necessary until only a small amount remains. Early needling, especially during the third or fourth

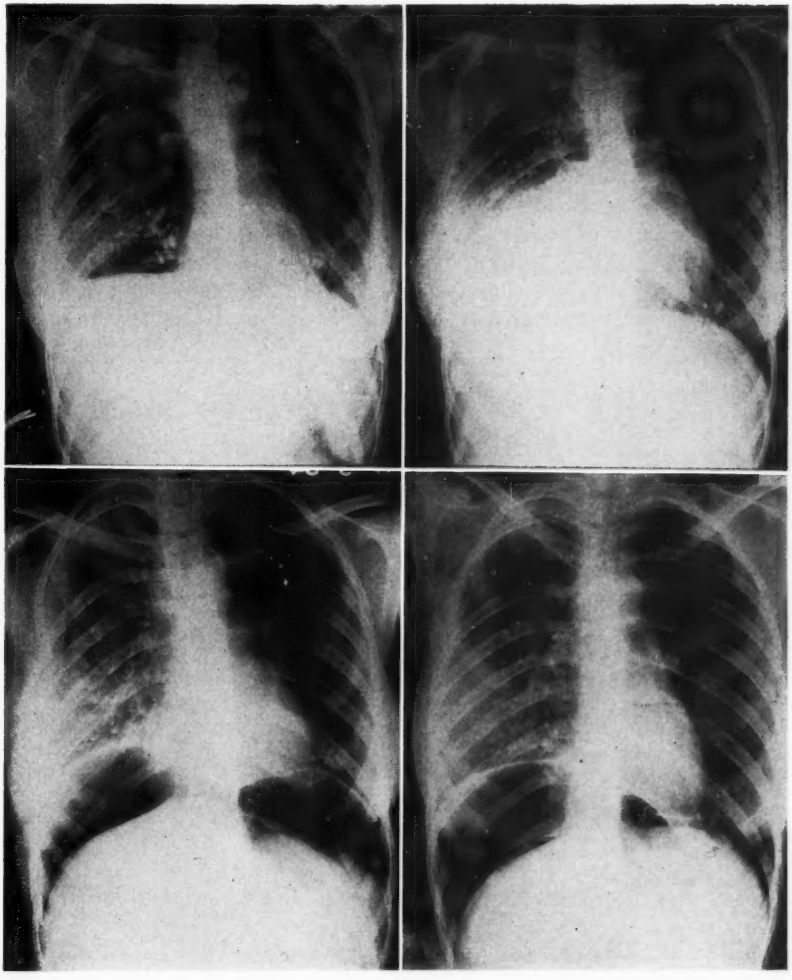


Fig. 4.—*A* (Nov. 5, 1954). On Oct. 31, 1954, the patient had an apicoposterior segmental resection of the right upper lobe for tuberculosis. By the fourth day after surgery, both upper and lower tubes were plugged and there was an apical space. The lower tube was removed. On Nov. 5, 1954, besides the apical space there was a paramediastinal air-fluid collection above the diaphragm. SK-SD was instilled through the upper tube. Some of the fluid and air was aspirated through the tube by high suction, but the fluid collection in the lower chest could not be removed by thoracentesis. This complication might have been avoided if the lower tube had not been removed.

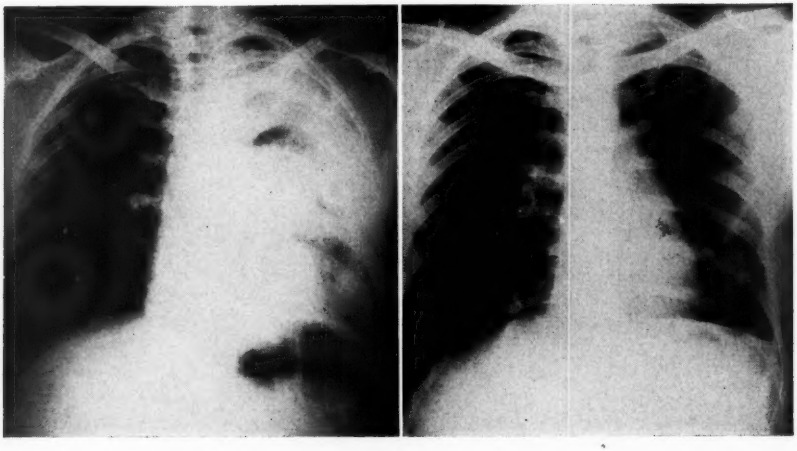
B (Nov. 7, 1954). On Nov. 7, 1954, there was a large supradiaphragmatic and paramediastinal collection. The apical air space was still present. On the same day a tube was inserted through the bed of the seventh rib to the paramediastinal space, and the apical space was intubated again. Pneumoperitoneum was started.

C (Nov. 9, 1954). Roentgenograms taken two days later showed marked improvement.

D (Dec. 10, 1954). Forty days after surgery, the pleural reaction had almost disappeared.

postoperative day, gives the best results. Once clotting occurs, several installations of SK-SD solution through one or more chest tubes have been beneficial (Fig. 5). There must be at least one tube in a dependent intrapleural position. Once fibroplasia has started, disappointing results are seen with this agent. It is best, therefore, to use it during the first postoperative week. The ideal result must show the entire superior surface of the diaphragm and completely expanded lung by roentgenography.

Occasionally, none of these procedures gives a good result. To prevent complications arising from a significant hemothorax, it is sometimes necessary to re-enter the chest through a short incision, evacuate all clots and fluid, and reinstitute closed intrapleural drainage. It is frequently easier to obtain a good result by this procedure than by decortication later. If evacuation is done before ten postoperative days have passed, a restricting visceral peel has not developed.



A. *B.*

Fig. 5.—*A* (March 29, 1954). On March 26, 1954, the patient had an apicoposterior segmental resection of the left upper lobe for tuberculosis. The pleural space was obliterated by adhesions and an extensive dissection was necessary to free the lung. Postoperatively there was considerable bleeding into the left chest. The chest film was as shown three days after operation. With both drainage tubes in place, SK-SD, tracheal suction, and intermittent positive pressure breathing were used repeatedly with gratifying results.

B (Aug. 11, 1954). Four and one-half months later the above roentgenogram was taken.

There is an ominous type of hemothorax that usually occurs during the first twenty-four hours after surgery. Persistent bleeding into the pleural space from a small vessel or vessels may lead to early clotting. The drainage of blood from the chest may be alarming but incomplete, and the problem of blood replacement becomes acute. The blood pressure becomes unstable despite repeated transfusions. The lung remnant is compressed and the mediastinum may shift to the opposite side. Changing the patient's position causes sudden hypotension. Before this clinical picture develops completely, re-exploration is mandatory with the evacuation of pleural contents and the ligation of bleeders.

THE APPEARANCE OF LATE PLEURAL SPACES AFTER RESECTION

Pneumothoraces may appear ten or more days after surgery. Some are seen as better roentgenograms or different projections are obtained. Although early chest films may have showed complete expansion, a thin air space was probably present. At any rate, the sudden appearance of a large space indicates a bronchopleural fistula, and prompt closed intubation should be done. Low suction to the drainage tube usually leads to pleural obliteration and closure of the fistula (Fig. 6).

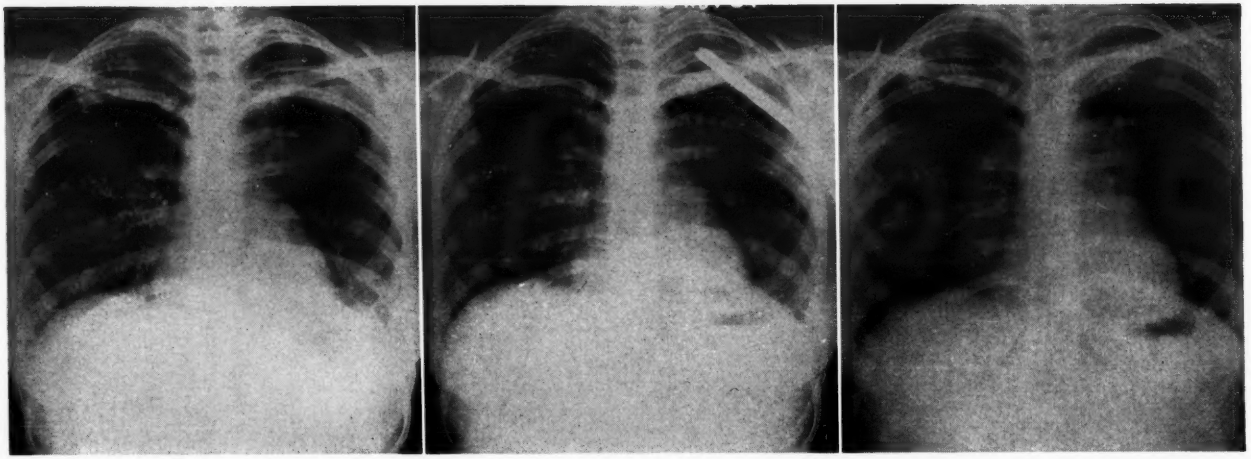
MANAGEMENT OF PERSISTENT AIR SPACES DESPITE ABOVE TREATMENT

A large air space that persists without improvement over two weeks after surgery often implies inadequate treatment earlier. If antibioties have been used, empyema usually has not developed even at this interval. At least systemic signs and symptoms of pleural infection are rare. The surgeon knows, however, that empyema is a constant threat, and he must get rid of this space as soon as possible. The procedures that must be used are more extensive than those previously mentioned. Generally, the longer necessary surgery is delayed, the greater will be the ultimate loss of pulmonary function.

When an air space persists without leakage from the lung remnant, it usually is situated in the upper half of the chest. With needle aspirations, high persistent negative pressures can be obtained. The upper portion of the lung, however, may not expand appreciably because there is a limiting visceral peel or there is disease within the lung remnant. An extensive and thick peel over the visceral pleura must be decorticated if the underlying lung is to increase in volume, and it may be necessary to excise the parietal surface of the space as well. If it is inadvisable to open the chest, a modified upper thoracoplasty may be beneficial. Frequently this is a safer operation. The number of ribs to be removed will depend on the size of the space to be closed. Usually a first-stage procedure suffices, and in the absence of a fistula, the space may be aspirated by needle through the parietal wall at the time of surgery.

When an apical space of any size persists due to a bronchopleural fistula, it must be intubated with the application of low suction (Fig. 7). The tube is best inserted in the third interspace as far to the front as possible but lateral to the pectoral muscle. After such drainage has been established, a modified thoracoplasty should be done taking care to avoid the tube tract. All efforts should be made to avoid contamination of the subscapular space. Again a first-stage operation usually is adequate, but on occasions the posterior segments of more ribs must be removed later. The establishment of drainage and thoracoplasty can be done concomitantly, but in the order mentioned. This surgery should not be put off too long, for progressive thickening of the parietal pleura may eventually prevent good collapse.

Persistent air spaces with bronchopleural fistulas in the lower chest that have received some treatment are usually not large. If they are to be eliminated, intubation with suction is necessary. The diaphragm should be elevated as high as possible by pneumoperitoneum. Sometimes a very small space



A. *B.* *C.*

Fig. 6.—*A* (Nov. 8, 1954). On May 14, 1954, the patient had an apicoposterior segmental resection of the right upper lobe for tuberculosis and she made a good recovery. On Oct. 6, 1954, the same operation was done on the left upper lobe. There was a space problem during the early postoperative course requiring needle aspiration and secondary intubation. The left pleural cavity seemed to be obliterated until four weeks after surgery when an apical space appeared. On Nov. 8, 1954, there was an air-fluid level in the left upper chest which could not be remedied by tapping although a fistula was not apparent by needle aspiration.

B (Nov. 16, 1954). A catheter was inserted into the space under direct vision through the anterior axilla with good expansion of the lung remnant by Nov. 16, 1954.

C (Nov. 27, 1954). By Nov. 27, 1954, the remaining left lung had expanded well into the apex.

A. *B.* *C.*

Fig. 7.—*A* (July 15, 1954). On June 30, 1954, the patient had an apicoposterior segmental resection for tuberculosis. At first her roentgenograms looked well but she later developed an apical space with evidence of a small fistula. Repeated needle aspirations were of no avail.

B (Aug. 17, 1954). On July 27, 1954, a modified right upper thoracoplasty was performed following intubation of the space. The space and fistula were closed without recurrence.

C (Dec. 10, 1954). Twelve weeks after resection her chest film is as shown.

still remains, usually because of leakage about the tube. It may then be wise to divide the tube at skin level and resort to open drainage. The residual pleural cavity is allowed to contract with repeated changes to smaller drainage tubes. Ultimate obliteration of space and fistula may occur. When this does not happen, the resection of more lung to include the fistula may be necessary after several months have elapsed.

SUMMARY

A persistent pleural space following partial resection of the lung may lead to empyema and bronchopleural fistula sometimes long after resective surgery. Prompt care must be given to obliterate the space soon after resection. Infection is usually absent at this time. The visceral pleura is pliable, and transected lung surfaces expand more readily. When this golden period of management is allowed to slip by and a pleural space persists, various processes hinder a satisfactory outcome. The depleuralized surface of lung and the visceral pleura become leathery and thick, impeding inflation of the lung remnant. Fistulous openings become more rigid, and they may not close when brought against another surface. Persistent air leaks maintain neutral or positive intrapleural pressures and hinder pulmonary expansion further. The hazard of infection increases daily.

Presented here are methods of management of the postoperative pleural space with illustrative case reports. Although therapeutic generalities are discussed, each patient must be treated according to his own local and general problems.

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FOREIGN-BODY BRONCHIECTASIS

JACK C. COOLEY, M.D.,* ROBERT L. GINSBERG, M.D.,* ARTHUR M. OLSEN, M.D.,** AND JOHN W. KIRKLIN, M.D.***
ROCHESTER, MINN.

DURING a recent review of the experience of the Mayo Clinic in bronchiectasis, it appeared that bronchial disease secondary to foreign bodies manifests certain different clinical and prognostic characteristics. It was with this observation in mind that separate consideration was given to foreign-body bronchiectasis.

MATERIALS AND METHODS

This study consists of 14 cases in which bronchiectasis apparently was caused by an obstruction of the bronchus and was secondary to a foreign body. The 14 patients were treated surgically from 1940 through 1953. Bronchoscopic, bronchographic, and pathologic studies were made in each case. Follow-up studies were possible in all 14 cases. Pulmonary resection was carried out in all instances with the aid of general endotracheal anesthesia, and the procedure consisted of a posterolateral thoracotomy incision and individual ligation of hilar structures. Pathologic examination confirmed the clinical impression of bronchiectasis in all cases, and in each case revealed the foreign body.

CLINICAL FEATURES

The distribution of these 14 patients according to age is given in Table I. Most of them were in the younger age groups. There were 8 males and 6 females

TABLE I. INCIDENCE ACCORDING TO AGE: 14 PATIENTS WHO HAD FOREIGN-BODY BRONCHIECTASIS

AGE (YEARS)	PATIENTS (NO.)
0-9	1
10-19	5
20-29	5
30-39	1
40-49	2

in the series. All the patients manifested chronic productive cough. The occurrence of hemoptysis (8 patients or 57 per cent) was in accordance with the findings of others reporting cases of foreign-body bronchiectasis.[1, 5] The interval after aspiration of the foreign body ranged between six months and

The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.
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*Fellow in Surgery, Mayo Foundation, Rochester, Minn.
**Section of Medicine, Mayo Clinic and Mayo Foundation, Rochester, Minn.
***Section of Surgery, Mayo Clinic and Mayo Foundation, Rochester, Minn.

twenty years. The exact distribution of this interval is given in Table II. The location of the foreign body was equally divided between the two sides, there being seven foreign bodies on each side.

TABLE II. INTERVAL BETWEEN ASPIRATION OF FOREIGN BODY AND OPERATION: 14 PATIENTS

INTERVAL (YEARS)	PATIENTS (NO.)
0-5	3
6-10	1
11-15	6
16-20	3
Not known	1

The lower lobe of the lung was involved in each case. No isolated instance of involvement of the upper lobe was found; however, in 5 cases involvement of the upper lobe accompanied involvement of other lobes of the lung, thus requiring pneumonectomy. In the remaining cases, unilateral unilobar resection was done in 8 of them, and unilateral multilobar resection (right middle and lower lobes) was done in 1.

RESULTS

There were no surgical deaths. An excellent result was obtained in every case. We regard an "excellent result" as one in which the patient has little if any residual cough and expectoration, and has obtained full functional rehabilitation.

Results of our study are in striking contrast with the clinical results obtained for 221 patients with primary bronchiectasis treated surgically at the Mayo Clinic during the same period; in that series an excellent result was obtained for 75 per cent of the patients.[2] Of the 18 patients in the group with primary bronchiectasis who underwent pneumonectomy, 14 (78 per cent) obtained a good result. This higher incidence of excellent results obtained for those who underwent pneumonectomy for primary bronchiectasis may partially explain why surgery produced such an excellent result for our patients with foreign-body bronchiectasis, since 5 of the 14 in this group underwent pneumonectomy. Furthermore, at operation it was noted that 6 of the patients with foreign-body bronchiectasis had marked contraction of the affected lobes. It is probable that the patients in this group already had made a physiologic adjustment to the loss of pulmonary tissue, and this may be another explanation of the good results obtained from surgery.

The postoperative complications were five instances of empyema and of hemothorax requiring two thoracenteses. It is interesting and evident that a good result may be obtained despite the occurrence of postoperative empyema.

There was no instance of postoperative atelectasis in these 14 cases, nor was there any retention of secretions sufficient to require bronchoscopy. Here, again, results were in striking contrast to those in the series of primary bronchiectasis[2] in which 25.8 per cent of the patients required bronchoscopy for retention of secretions.

CONCLUSIONS

The prognosis after pulmonary resection for foreign-body bronchiectasis is good. An excellent clinical result was obtained for all patients with foreign-body bronchiectasis treated surgically, as opposed to excellent results obtained for only 75 per cent of patients who had primary bronchiectasis. Postoperative atelectasis, occurring in 25.8 per cent of patients who had primary bronchiectasis, did not appear in 14 who had foreign-body bronchiectasis. By contrast, the incidence of postoperative empyema among those who had foreign-body bronchiectasis was much more (30 per cent) than it was among those who had primary bronchiectasis (6 per cent). We attribute this difference to the increase in obstructive pneumonitis in the presence of foreign-body bronchiectasis.

On the basis of the foregoing data, it is apparent that the prognosis after surgical resection for foreign-body bronchiectasis is much better than it is after surgical resection for the primary form of the disease. This would seem to indicate that there are basic pathogenetic and physiologic, if not pathologic, differences inherent in foreign-body bronchiectasis that distinguish it from primary bronchiectasis. Early bronchoscopic aspiration of foreign bodies is important in the prophylaxis of foreign-body bronchiectasis,[3, 4] whereas surgical resection would appear to be the treatment of choice when the condition has already developed.

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EXPERIMENTAL REPLACEMENT OF THE SUPERIOR VENA CAVA WITH HOMOLOGOUS ARTERIES, AND REPORT OF A CASE WITH MALIGNANT OBSTRUCTION REPLACED WITH A HETEROLOGOUS ARTERY

FRANK S. ASHBURN, M.D., WILLIAM H. SEWELL, LIEUTENANT (MC) USNR, AND CHARLES E. HUGGINS, LIEUTENANT (MC) USNR
WASHINGTON, D. C., AND BETHESDA, MD.

INTRODUCTION

ACUTE obstruction of the superior vena cava, though infrequently encountered, is of clinical importance because of the discomfort and incapacitating effects on the patient. In a review of the subject,[1] malignancy was reported to have caused 44 per cent of the obstructions; aortic aneurysm, 32 per cent; and undetermined origin, believed to be a form of mediastinitis, 12 per cent. In another series,[2] signs and symptoms of superior caval obstruction were present on admission in 2.2 per cent of patients with proved carcinoma of the lung.

There have been occasional reports of surgical attempts to relieve the obstruction.[3-9] A patient with superior caval obstruction caused by a malignancy may be treated by external radiation. Relief of the symptoms is often obtained. If surgical exploration is indicated for possible palliative resection of the lung, and if technical factors are favorable for resection and grafting, removal of the involved vessel and replacement with a graft now appears to be the procedure of choice. Immediate relief of symptoms can be expected and there is the likelihood of making the patient more comfortable for a significantly larger percentage of his life expectancy.

Good results have been reported with autogenous femoral vein grafts used for the replacement of the superior cava.[6-8] Both viable and nonviable homologous arteries have also been used for this purpose with success.[7-9] Homografts seem to be more likely to succeed when preserved by the freeze-drying process than when in the viable state, judging from small differences observed between the results of series of freeze-dried and comparable fresh homografts in the abdominal aortas of dogs.[10, 11]

Autogenous vein is probably the material of choice for caval replacement. The additional incision required is a disadvantage. Extensive studies have not been done to determine the relative incidence of thromboses in autogenous vein grafts versus homologous arteries implanted in veins. However, a greater

From Thoracic Surgical Service, Doctors Hospital, Washington, D. C., and Naval Medical Research Institute, National Naval Medical Center, Bethesda, Md.

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tendency toward thrombosis has been demonstrated in freeze-dried homologous arteries than in free autogenous carotid grafts in the dog.[12] This is a region where the tendency to thrombosis in homografts is high because of the small size of the vessel.[12] Minimal foreign tissue reaction may cause an occlusive thrombus in a small vessel or one in which flow is slow.

Freeze-dried arterial heterografts offer another possible solution to the problem. They have been found to be more successful than fresh heterografts when used in the abdominal aortas of dogs. A freeze-dried vessel contains less soluble protein material than a fresh unextracted one.[13, 14] Successful results using freeze-dried bovine and porcine heterografts in a short series of human cases were reported by Hufnagel.[15]

While some woven plastic prostheses are satisfactory in large- and medium-sized arteries, experimental work in animals has made them appear extremely unpromising for the superior vena cava.[16] Though shunts are useful, and probably essential in some locations, to maintain circulation during the resection and grafting of certain segments of the aorta,[17, 18] there appears to be no indication for the use of a shunt around a vein which is largely or completely obstructed at the time of exploration.

Animal experiments have been undertaken to study the use of freeze-dried homologous arterial grafts in the superior vena cava.

METHOD

The descending thoracic aorta was removed from 45- to 60-pound dogs, using sterile autopsy technique. This tissue was freeze-dried according to the method of Pate.[10]

Right thoracotomy using aseptic technique was performed on dogs under intravenous Nembutal anesthesia and 100 per cent endotracheal oxygen under intermittent positive pressure. The superior vena cava was occluded at its origin with a rubber-shod Rankin hemostat and the pressure on the vessel controlled by a rubber band around the handle. A V-shaped portion of the right superior aspect of the right auricle, near the base of the auricular appendage, was occluded with Potts' clamps. After the cava was resected, the graft was sutured first to the upper end of the cava using two stay sutures, connected by continuous everting mattress sutures of No. 5-0 silk. The graft was then sutured to the auricle in like manner, with careful eversion of the auricular edges. No anticoagulants or antibiotics were used postoperatively.

RESULTS

There were no early postoperative deaths. Of the 6 dogs operated on, 1 died after four weeks. At autopsy, the pleural cavity was found to be largely filled with fluid, and the graft was completely thrombosed. The other 5 dogs were sacrificed after 1, 1½, 4, 5, and 5 months. In all cases, each graft was the same size as when implanted, with no evidence of excessive fibrosis around the outside or mural thrombus on the inside. The suture line between the superior vena cava and the graft was well endothelized and without stricture. How-

ever, the anastomosis between the graft and the right auricle was found to contain a thin diaphragmlike stricture in all cases. The opening was only one-third to one-fourth the original diameter of the graft. The stricture appeared to have formed in the auricle immediately adjacent to the anastomosis and may have been the result of an organized thrombus.

Cephalad venous pressure was elevated in all cases after approximately two weeks, and significant pleural effusion was found at autopsy in 2 of the dogs. Microscopic examination of the grafts showed good endothelization, loss of the muscular elements with persistence of the elastic fibers, and invasion by host fibroblasts. This picture was similar to that obtained with freeze-dried homografts in the abdominal aorta.

Fig. 1.—Specimen from a dog sacrificed five months after replacement of superior vena cava by a freeze-dried aortic homograft. The condition of the graft and upper suture line is good, but there is severe stricture at the graft to right auricular anastomosis.

DISCUSSION

These results would seem to suggest that a graft to cava anastomosis is likely to succeed, whereas one between the graft and the auricle is likely to stricture. These strictures at the auricular anastomosis are similar to those noted in some cases by Gerbode[19] and others[7] after transplantation of vessels to the right auricle.

The first successful treatment of superior vena caval occlusion by arterial graft was reported by Holman and Steinberg in 1954.[9] In their case, occlusion

was caused by nonspecific mediastinitis and the occlusive process involved the entire vena cava save a small segment at either extremity, thereby enabling them to use a freeze-dried arterial homograft in graft to cava anastomosis rather than graft to auricle. Their patient has done well since operation and certainly (to use the words of Holman and Steinberg[9]) "the desperate plight of patients with such an obstruction warrants continued trial of this procedure."

CASE REPORT

A case is reported illustrating the possibility of obtaining a successful result with a freeze-dried porcine aortic graft, when surgical replacement is desired and alternative procedures are not feasible for one reason or another.

This is the case of a 69-year-old white man, who was admitted to Doctors Hospital on March 26, 1955, complaining of swelling of the face, neck, arms, and chest, for one week. He stated that while on his yacht about a week prior to his admission, March 17, 1955, he awoke one morning to find his face markedly swollen and blue, as well as the arms and upper chest. The veins and venous pattern on the chest were very prominent and the veins in the neck stood out like large cords.

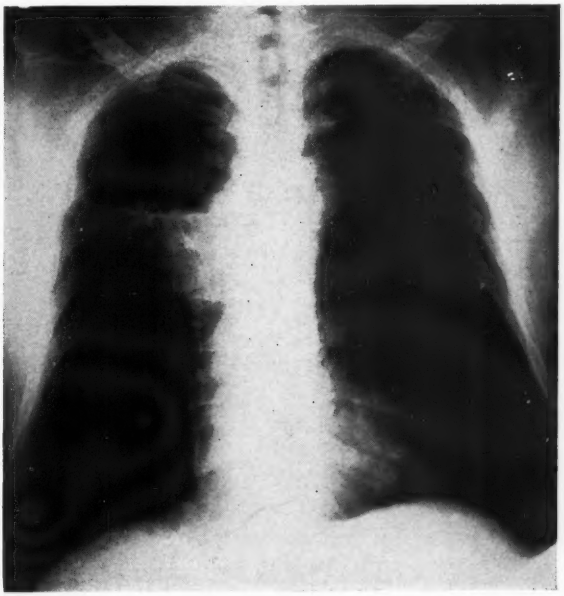

Fig. 2.—Preoperative chest x-rays showing lesion in the right hilum and the cyst noted previously in the right upper lobe.

For several years he had complained of severe cough. However, this had become more severe within the last six months, and at times he was seized with severe paroxysms lasting several minutes. He had never coughed up blood and was not hoarse, and he had no difficulty in swallowing. In the past, he had been seen repeatedly by his internist because of a chronic peptic ulcer, and chest x-rays made in 1950 and 1951, both showed a large cyst in the right upper lobe. He was acquainted with this fact but had had no treatment for this condition. His family history was nonrevealing.

At the time of his admission, he was an obese white man whose face and whole upper body were cyanotic with many prominent veins and venules present over the chest and back. The venous pressure was markedly elevated in the upper extremities, as evidenced by the veins in the hand which remained completely engorged even when the hands were elevated high above the head. He was most uncomfortable and felt the engorgement of his face and head very keenly, and was also somewhat dyspneic. The heart was slightly enlarged to the left and there was a systolic apical murmur. The percussion note over the right upper lobe was somewhat more resonant than normal; however, no dullness could be elicited. Moist râles were heard in the right lower chest. No breath sounds were heard in the right apex. There was no palpable adenopathy and examination of neither the abdomen nor the remainder of the body was remarkable. The blood pressure was 164/80, pulse, 82, and the temperature was 98.6° F.

An x-ray examination of the chest, including fluoroscopy and laminographic studies, showed the presence of a large soft tissue mass apparently arising in the mediastinum near the tracheal bifurcation and projecting into the upper half of the right lower lobe, partial collapse of the right lung, and moderately severe tortuosity of the aorta with calcification of the arch. The mass projecting into the right lower lobe was nonpulsatile on fluoroscopy. Barium swallow revealed localized posterior displacement of the esophagus on the posterior aspect of the heart consistent with enlargement of the left atrium.

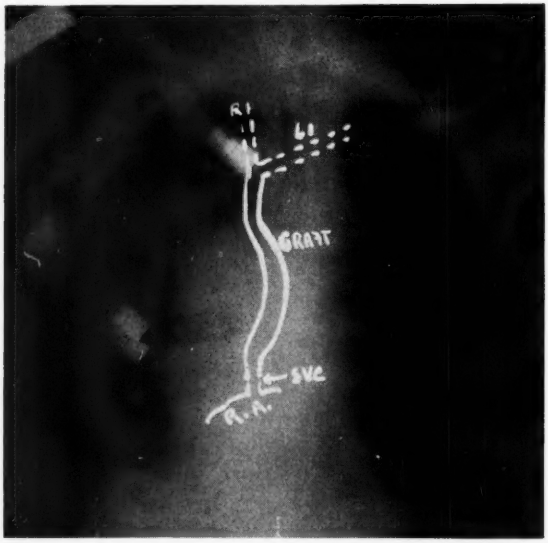

Fig. 3.—Postoperative film, with outline of graft indicated.

Laminographic studies contributed little other information except to indicate that there was widening of the right main-stem bronchus probably indicating pressure by the mass on the bronchus from behind. There was no evidence suggesting dilatation of the aorta.

On March 19, 1955, he was bronchoscoped, and it was noted that the trachea was fixed in the midline and the carina was greatly widened. Both main-stem bronchi were decreased in caliber; however, no gross tumor was visualized. Bronchial washings were made and sent to the laboratory for study. These washings failed to reveal contributory findings.

A diagnosis was made of bronchogenic carcinoma, probably not resectable, plus a complete block of the superior vena cava. On March 23, 1955, a right exploratory thoracotomy

was done and a large mass was found in the right hilum, completely occluding the superior vena cava and surrounding both bronchi. Frozen sections made from the lesion revealed anaplastic carcinoma, probably of bronchogenic origin.

The superior vena cava was involved in this mass almost throughout the extent of the vessel. At its origin, from the innominate veins, it was possible to dissect a segment of uninvolved cava for about 1.5 cm. This gave an adequate cuff for anastomosis at this site. The growth involved the site of entrance of the azygos vein into the superior vena cava as well as the pericardium at the point of entrance of the superior vena cava into the pericardial sac. Fortunately, however, when the pericardium was opened it was possible to dissect a cuff of vena cava approximately 1 cm. in length, which proved adequate for anastomosis of the proximal end of the graft. Noncrushing clamps were used to occlude the cava at both extremities and the vessel was divided first at its upper extremity. A suitable length of reconstituted freeze-dried porcine aorta was selected for use as the graft to bridge the defect in the cava, and the anastomosis was accomplished by continuous everting mattress suture of No. 5-0 silk. The intrapericardial portion of the cava was divided and the remaining end of the graft was sutured to the auricular stump of the superior vena cava by the same technique. No clamp was applied to the graft at any time. The remaining cut ends of the involved cava did not bleed and the involvement was so extensive that no attempt at resection of the mass or vena cava was attempted. The uppermost clamp was removed first and as the graft filled with blood, the air it contained was displaced through the suture lines; however, very little blood leaked through and this soon ceased completely.

When the lower occluding clamp was removed, adequate blood flow occurred through the graft and the anesthetist reported prompt improvement in the color of the patient's face and neck. The following day, the patient's edema and abnormal venous pattern over the upper body had disappeared. He left the hospital on the seventh postoperative day and has been undergoing roentgen therapy since his discharge. He has returned to work, and has had no recurrence of swelling of the face and upper body seven months after operation.

SUMMARY AND CONCLUSIONS

1. Superior vena cava obstruction caused by malignant invasion is infrequent but constitutes a definite clinical problem.

2. Radiation therapy is often satisfactory and is advised if palliative resection is not desired.

3. Freeze-dried arterial homografts were used experimentally for replacement of the superior vena cava in 6 dogs. The cava to graft anastomosis and the graft itself were successful, but the graft to right auricular anastomosis resulted in stricture.

4. A case is reported in which a freeze-dried porcine aortic graft was sutured to stumps of the superior vena cava at each end with a satisfactory result.

5. Resection and replacement by a graft, of a cava obstructed by malignant disease, is advised if exploration is indicated for possible resection of the primary tumor, and the graft can be sutured to vein at each end.

6. Indications for grafting are less definite when exploration is not otherwise indicated, or when the graft can be sutured only to the right auricle proximally.

7. Materials which can be used with success for replacement of the resected cava, in descending order of preference, are autogenous femoral vein, freeze-dried homologous artery, and freeze-dried porcine aorta.

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POSTOPERATIVE COMPLICATIONS RELATED TO PREOPERATIVE ANTIMICROBIAL THERAPY AND OTHER FACTORS

A Review of 600 Consecutive Pulmonary Resections for Tuberculosis*

J. E. Hiltz, M.D., J. J. Quinlan, M.D., and V. D. Schaffner, M.D.
Kentville, Nova Scotia

OVER the years since 1944, when the first pulmonary resection for tuberculosis was done at the Nova Scotia Sanatorium, it has been intriguing to speculate upon the reason why one patient would encounter a postoperative complication whereas some other, apparently similar person, would not. Recently there have been a few scientific papers presented suggesting that prolonged preoperative antimicrobial therapy has led to the development of drug resistance in many of the patients and so is responsible for ensuing complications.

In an attempt to evaluate the situation as it exists at the Nova Scotia Sanatorium, it was determined to analyze the first 600 pulmonary resections for tuberculosis done there between Nov. 14, 1944, and March 25, 1955.

As postoperative complications of resection, the following were accepted: (a) early bronchopleural fistula (within two weeks of operation), (b) late bronchopleural fistula, (c) empyema, (d) spread of disease (contralateral and homolateral), (e) persisting pleural space, (f) massive wound disruption (2 cases only without above-mentioned additional complications), and (g) early death (2 cases only; cause unknown).

Table I indicates the age groups and sex of all patients and also of those who developed any of the complications just mentioned. It had previously been noted, but not confirmed statistically, that complications were much more

Table I. Age Groups and Sex of All Patients Undergoing Resection With Age, Sex, and Percentage of Each Sex Group Developing Postoperative Complications

Age Groups*	All Cases			Cases With Postoperative Complications					
	Male	Female	Both Sexes	Male		Female		Both Sexes	
	Number	Number	Number	No.	%	No.	%	No.	%
0- 9	3	1	4	0	–	1	100	1	25
10-19	19	47	66	6	31	8	17	14	21
20-29	82	159	241	23	28	30	19	53	22
30-39	59	99	158	18	30	22	22	40	25
40-49	64	47	111	27	42	7	15	34	31
50-59	11	8	19	7	64	1	12	8	42
60-69	1	0	1	0	–	0	–	0	–
All ages	239	361	600	81	34	69	19	150	25

*Youngest; 7 years, 10 months: oldest; 64 years, 10 months.

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*From Nova Scotia Sanatorium, Kentville, Nova Scotia.

likely to occur in men than women; in this series they occurred in 34 per cent of the men as compared to 19 per cent of the women, in spite of the fact that almost 50 per cent more women than men underwent pulmonary resection. Although the numbers are rather small, the tables would also seem to suggest that men in the older age groups, 40 to 49 years and more especially 50 to 59 years, were more prone to complications. When the sexes were combined, the complication rate was roughly 25 per cent, increasing over the age of 40 years, the increase being due to the male patients alone.

The next problem was to attempt to determine if the extent or magnitude of the operation influenced the preponderance of complications postoperatively.

Table II would suggest that the greater the amount of lung tissue removed, the greater the chances of developing postoperative complications, there being 43 per cent complications among the pneumonectomy group, but only 3 per cent among those undergoing wedge resections. The lobectomies represented the average for all operations, 25 per cent. In some of the groups the numbers of operations done were too small to be significant.

TABLE II. THE TYPE OF OPERATION PERFORMED RELATED TO POSTOPERATIVE COMPLICATIONS

TYPE OF OPERATION	NUMBER DONE	ALL COMPLICATIONS		EARLY FISTULA	LATE FISTULA	EMPYEMA	SPREAD OF DISEASE	STUBBORN SPACES
		NO.	%	NUMBER	NUMBER	NUMBER	NUMBER	NUMBER
Pneumonectomy	92	40	43	24	2	23	17	24
Lobectomy	280	69	25	41	0	38	24	44
Bilobectomy	20	6	30	1	1	3	2	3
Lobe + segment	39	16	41	14	0	12	3	11
Lobe + wedge	10	1	10	0	0	0	0	1
Lobe + segment + wedge	2	1	50	1	0	1	0	1
Segmental resection	48	11	23	6	0	4	2	8
Segment + wedge	3	1	33	1	0	1	0	0
Multiple wedge	9	2	22	0	0	0	2	0
Wedge only	97	3	3	1	0	2	1	1
All cases	600	150	25	89	3	84	51	93

Complications, of course, were frequently multiple, bronchopleural fistula often being followed by or preceded by empyema.

The presence of bronchial tuberculosis would be expected to influence the development of postoperative complications, especially bronchopleural fistula. As a matter of policy, no patient was passed for surgery before bronchoscopy had been carried out. If a definite active endobronchial tuberculosis was discovered, antimicrobial therapy was continued until subsequent bronchoscopic examination revealed that the process had healed. Unfortunately, a mucosal lesion may heal very much more quickly than the submucosal disease, but there is no way to determine the state of the submucosal tissues. During the later years of resectional surgery here, the surgeon made special notes and the pathologist reported especially upon the state of the resected bronchial tree where a major bronchus was severed.

Table III presents information about the relationship of tuberculous tracheobronchitis and postoperative complications. Thirty-two patients who had

evidence of gross tuberculous tracheobronchitis at the time of operation, and 33 persons with microscopic evidence of active endobronchial disease, and 44 who had shown a prior lesion bronchoscopically, did not develop any postoperative complications. Evidently, therefore, this preoperative condition need not necessarily lead to postoperative disaster, especially if re-expansion of remaining lung tissue may be brought about promptly to cover the stump and if surgery is done under adequate antimicrobial cover. It is of interest, however, and shown by this table that of the patients reported as having gross or microscopic endobronchial disease at the time of operation, the postoperative complication rate was definitely and appreciably increased, 36 to 39 per cent of cases, whereas when no endobronchial disease was seen, the complication rate was a surprisingly uniform average of 26 and 24 per cent.

TABLE III. RELATIONSHIP OF TUBERCULOUS TRACHEOBRONCHITIS TO THE DEVELOPMENT OF POSTOPERATIVE COMPLICATIONS*

TUBERCULOUS TRACHEOBRONCHITIS	NUMBER DONE	NO COMPLICATIONS NUMBER	ALL COMPLICATIONS NO.	ALL COMPLICATIONS %	EARLY FISTULA	LATE FISTULA	EMPYEMA	SPREAD OF DISEASE	STUBBORN SPACE
a									
Gross	50	32	18	36	14	0	14	3	13
None seen	299	220	79	26	47	2	39	25	52
Not stated	251	198	53		28	1	31	23	28
b									
Microscopic	54	33	21	39	15	0	13	5	12
None microscopically	214	162	52	24	31	1	28	12	34
Round-cell infiltration	58	44	14	24	11	0	8	3	11
Not stated	274	211	63		32	2	35	31	36
c									
Bronchoscopically seen before	54	44	10	19	6	0	8	2	6
None seen	530	399	131	25	79	2	70	43	81
Not stated	16	7	9		4	1	6	6	6

*Sections *a*, *b*, and *c* are not self excluding but show a considerable overlap.

When endobronchial disease had been noted bronchoscopically, the complication rate was below average (19 per cent) which may well be attributed to the prolonged antimicrobial treatment given for this condition preoperatively.

Antimicrobial therapy was first administered at the Sanatorium in the form of streptomycin in February, 1947, followed by PAS in August, 1949, thiosemicarbazone in September, 1950, and isoniazid in June, 1952. During the early years, of necessity, a number of our patients did not receive any antimicrobial therapy pre- or postoperatively. This was followed by a period when patients received streptomycin alone. As soon as a second drug became available, however, it was our policy to administer at least two of the available drugs in combination at all times even to the point of using thiosemicarbazone more frequently than its therapeutic worth by itself might have warranted.

As was to be expected, 52 per cent of all patients who did not receive preoperative chemotherapy developed one or more of the major complications in the postoperative period. This is shown in Table IV. As was anticipated, also,

those patients who were able to receive only one drug preoperatively developed fewer complications if the drug was administered for less than three months preoperatively than if given for longer periods, 26 per cent as compared to 50 to 55 per cent. In other words, those patients who received only small quantities of a single drug preoperatively were less likely to develop resistance and so were able to benefit from it for longer periods of time postoperatively. Most of the patients who received a single drug for less than three months did so for approximately three to seven days only. Many of these patients, too, were in a more nearly arrested stage of the disease at the time of operation, being early cases in this resection series. They had reached the so-called "target point" without benefit of antimicrobial therapy.

TABLE IV. COMPLICATIONS RELATED TO THE ADMINISTRATION OF NO PREOPERATIVE ANTIMICROBIAL DRUGS OR A SINGLE DRUG FOR VARIOUS PERIODS OF PREOPERATIVE TIME

	NO PREOPERATIVE DRUGS		ONE DRUG ONLY PREOPERATIVELY													
			<3 MOS.		3-6 MONTHS		6-12 MONTHS		12-18 MONTHS		1½-2 YEARS		2 YEARS +		TOTAL	
	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%
Complications	11	52	9	26	6	55	1	50	1	100	0	—	0	—	17	35
No complications	10	48	25	74	5	45	1	50	0	—	1	100	0	—	32	65
Total	21	100	34	100	11	100	2	100	1	100	1	100	0	—	49	100

Table V shows the number and the percentage of patients who developed complications depending upon the type of operation performed and the duration of antimicrobial therapy. The figures for pneumonectomies, lobectomies, and wedge resections would seem to be large enough to be significant.

As was to be expected, where no preoperative chemotherapy was given, the complication rate was high, approximately 53 per cent. It is to be remarked, however, that most of these patients did not have postoperative antimicrobial therapy either.

As therapy with a single drug is no longer necessary or favored, it seemed well to present the picture after the exclusion of the cases which did not receive any antimicrobial drugs or received only one. This is shown in Table VI as it applies to all types of operations.

Actually, there is very little variation in the percentage of patients developing postoperative complications regardless of the shortness or length of time of the preoperative medication. There was a tendency, however, for those patients who received antimicrobial treatment for less than three months to develop more complications than those receiving the drugs from three months to over a year and a half (28 per cent as compared to 19 to 24 per cent). One can only assume that the active component of the disease was not sufficiently controlled in these cases preoperatively.

By the same token, those that received combined therapy preoperatively for a period of over two years had a higher complication rate, also 28 per cent. It is to be remembered, however, that they had received combined therapy for this prolonged period because the seriousness and extent of the disease warranted

TABLE V. COMPLICATIONS RELATED TO TYPE OF OPERATION AND LENGTH OF ANTIMICROBIAL THERAPY SHOWN

OPERATION		COMPLICATIONS SHOWN ACCORDING TO LENGTH OF PREOPERATIVE CHEMOTHERAPY															
		NO CHEMOTHERAPY		<3 MOS.		3-6 MONTHS		6-12 MONTHS		1-1½ YEARS		1½-2 YEARS		2 YEARS +		TOTAL COMPLICATIONS	
TYPE OF OPERATION	NO.	NUMBER	%	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%	NUMBER	%
Pneumonectomy	92	3	60	14	46	8	40	3	30	6	50	3	50	3	33	40	44
Lobectomy	280	5	50	20	23	10	23	17	21	12	29	2	15	3	42	69	25
Bilobectomy	20	3	60	1	14	0	0	1	50	—	—	1	100	0	0	6	30
Lobe + segment	39	—	—	4	25	2	66	5	39	5	83	0	0	–	—	16	41
Lobe + wedge	10	—	—	0	0	0	0	1	25	0	0	–	—	–	—	1	10
Lobe + segment + wedge	2	—	—	1	100	0	0	—	—	—	—	–	—	–	—	1	50
Segmental	48	—	—	5	42	1	25	3	19	2	18	0	0	0	0	11	23
Segment + wedge	3	—	—	—	—	1	50	0	0	—	—	–	—	–	—	1	33
Multiple wedge	9	—	—	0	0	0	0	1	33	1	33	–	—	0	0	2	22
Wedge only	97	0	0	0	0	0	0	3	9	0	0	0	0	0	0	3	3
All cases	600	11	53	45	28	22	23	34	21	26	25	6	19	6	29	150	25

– No cases in group.
0 No complications among cases in group.

it. In most cases, surgery would not have been either reasonable or possible without this extensive period of preoperative preparation. The total complication rate was 23 per cent for all patients receiving two or more drugs preoperatively.

TABLE VI. COMBINED THERAPY ONLY. COMPLICATIONS RELATED TO DURATION OF TIME OF ANTIMICROBIAL THERAPY PREOPERATIVELY

	<3 MOS.	3-6 MONTHS	6-12 MONTHS	12-18 MONTHS	1½-2 YEARS	2 YEARS +	TOTAL
All cases on combined therapy	128	85	162	103	31	21	530
Number with complications	36	16	33	25	6	6	122
Per cent with complications	28	19	20	24	19	28	23

As deaths reflect to some extent the complication rate, it may be of interest to show (Table VII) that the pneumonectomy operations carried the highest operative and over-all mortality, 8.7 per cent and 20 per cent, respectively. The operative mortality for all cases, 1944 to 1955, was 4.3 per cent within sixty days of operation and 7.8 per cent for all causes from three months to eleven years postoperatively.

TABLE VII. DEATHS, ANY CAUSE, ACCORDING TO TYPE OF OPERATION

TYPE OF OPERATION	NUMBER	OPERATIVE MORTALITY (WITHIN 60 DAYS)	PRESENT STATUS KNOWN	OVER-ALL MORTALITY TO APRIL 30, 1955
Pneumonectomy	92	8 or 8.7%	91	18 or 20%
Lobectomy	280	15 or 5.4%	273	24 or 8.8%
Bilobectomy	20	1	20	1
Lobectomy + segment	39	1	36	1
Segmentectomy	48	1	47	2
Wedge	97	0	96	0
Other	24	0	24	0
All cases	600	26 or 4.3%	587	46 or 7.8%

DISCUSSION

It is generally accepted that, where feasible, a pulmonary resection is the operation of choice for tuberculosis of the lung. In theory, at least, it removes the gross tuberculous lesion from the body. Unfortunately, unsuspected or microscopic areas of tuberculous disease are frequently left behind either within the lung parenchyma or in the bronchial walls.

It is suggested by the present analysis that men do not respond to this form of surgery as well as women and that, after the age of 40 years, the response becomes less favorable with the passage of time. Of the 15 men over the age of 40 years who had evidence of gross or microscopic endobronchial disease and underwent a pneumonectomy, or a lobectomy, or a lobectomy in combination with a more minor operation, 66 per cent developed complications. Of the remaining 49 men over 40 years of age without gross or microscopic endobronchial disease, 45 per cent developed complications compared to an average of 25 per cent for the whole group of resection cases. (Of the last 400 cases of this series, the complication rate was only 19.2 per cent, all having received com-

bined therapy.) The presence of previous or concurrent tuberculous endobronchial disease, known or unsuspected, increases the hazard of the operation, especially if the bronchial lesion occurs at the site of resection. It has been shown previously that ulcerative lesions of the bronchus will heal rapidly, often within one to three months,[1] following the initiation of antimicrobial therapy. Unfortunately, we lack similar knowledge regarding the rate of healing of submucosal tuberculous lesions which still present an operative hazard.

It would seem from the figures presented that there is some merit in trying to bring the patient to the so-called "target point" before undertaking surgery if by "target point" we may indicate relative stability of the lesion obtained by treating the patient with antimicrobial drugs sufficiently long to arrest submucosal tuberculosis as well as eliminate the exudative components of the parenchymal lesion. Less than three to six months antimicrobial therapy would seem to be inadequate. Three to eighteen months preoperative "combined drug" therapy, where indicated, would not seem to increase the risk of developing complications during the postoperative period.

Many of those patients who received drug therapy for over two years before operation did so because the disease was too extensive or too active to warrant resection sooner. The prolonged drug therapy brought them to a state of improvement in which surgery could be undertaken with relative safety. The prolonged period of preoperative treatment should not be blamed for causing postoperative complications. In many cases it was lifesaving and in 72 per cent of those who received drugs for over two years there were no complications.

The operative death rate in this series was 4.3 per cent with an over-all rate of 7.8 per cent. Excluding the pneumonectomies, the rate for the series is 3.5 per cent and 5.6 per cent, respectively, with no mortality for wedge resections. Undoubtedly, a pneumonectomy is the resection operation of the greatest magnitude. Not only does it carry with it the greatest surgical risk, but the patient is like a two-engine airplane carrying on with only one motor for the rest of his life. If anything, even trivial, happens to the remaining organ, the result may be catastrophic. Similarly, a single fuel pump, the heart, carries the responsibility for circulating the required vital fluid of the whole mechanism to the single motor. Even a minor failure here in the circulating mechanism will diminish its function below a life-sustaining level. There is an impression that cor pulmonale plays a larger part in our mortality figures than was at one time thought to be the case.

SUMMARY

1. Six hundred consecutive cases of pulmonary resection at the Nova Scotia Sanatorium have been reviewed.

2. The possible influence of age, sex, endobronchial tuberculosis, and the duration of preoperative antimicrobial therapy have been tabulated.

3. Mortality figures are given.

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INTRATHORACIC NEUROFIBROMA OF THE VAGUS NERVE

A Case Report

Wm. M. Tuttle, M.D., Detroit, Mich., V. Sanai, M.D., New Delhi, India, and H. P. Harms, M.D., Holland, Mich.
Detroit, Mich.

NEUROFIBROMAS arising in the posterior mediastinum are not uncommon, and at times they are associated without generalized neurofibromatosis (von Recklinghausen's disease). Such posterior mediastinal tumors have their origin from the intercostal nerves or the sympathetic chain. Neurofibromas of the cranial nerves are not common, the commonest nerve involved being the acoustic nerve.[1] Tumors of the cervical segment of the vagus nerve have been reported by a number of authors, but those arising in the thoracic segment are rare. Blades and Dugan[2] reported the first patient suffering from a thoracic neurofibroma of the left vagus nerve and its successful extirpation. Furrer and Fox[3] reported a patient with a perineural fibrosarcoma of the left vagus nerve which was discovered at post-mortem examination. Efskind and Liavaag[4] in their review of 21 cases of intrathoracic neurogenic tumors reported 1 patient with a neurinoma which had its origin in the recurrent laryngeal nerve. Recently Gerbode and Marguiles[5] reviewed the literature on this subject and reported 1 case of neurofibromatosis with neurofibromas of vagus nerve in the superior mediastinum associated with pectus excavatum. Since then Gilbertsen and Lillehei[6] have reported the instance of a patient with bilateral neurofibroma of the vagus nerve unassociated with generalized neurofibromatosis. It would appear that neurofibromas of the vagus nerve associated with generalized neurofibromatosis are uncommon. The present case report falls within this category. The tumor arose from the left vagus nerve immediately below the origin of the recurrent laryngeal nerve and was successfully extirpated.

CASE REPORT

S. W. (A-169238), a 30-year-old white man, was admitted to the Harper Hospital, Detroit, Mich., on Dec. 29, 1953, because of a mediastinal mass which was discovered on a routine yearly chest film. The patient was an employee of a public school system, and had had chest x-rays each year. He stated these chest examinations had been negative. The patient had no complaints or symptoms. There was no family history of tuberculosis, but the patient stated that he had had a positive tuberculin test for years. The past history revealed that he had had a toxic goiter as a boy, which was treated medically and later by x-ray. An appendectomy had been performed ten years before.

From the Department of Thoracic Surgery, Harper Hospital, Detroit, and the Department of Surgery, Wayne University College of Medicine, Detroit, Mich.
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On admission the physical examination revealed a well-developed, well-nourished, white man, appearing healthy, and in no apparent distress. The physical examination was entirely negative, and there was no evidence of any neurofibromatosis. The blood pressure was 140/90. The pulse was 74 per minute. The admission complete blood count, urine analysis, total serum proteins, and albumin-globulin ratio were within normal limits.

An x-ray of the chest revealed an oval soft tissue mass in the left mid-mediastinal area just above the left hilum. Fluoroscopically, the mass lay just above the main pulmonary artery on the left. No pulsations were observed in this area. The nature of this mass could not be determined by roentgenographic studies.

Fig. 1.—A posteroanterior projection of the chest reveals a tumor mass protruding from below the arch of the aorta, the nature of which was not determined until at the time of operation.

On Dec. 30, 1953, the patient was taken to the operating room and under intravenous Pentothal, curare, procaine, and intratracheal nitrous oxide and oxygen anesthesia with the patient in the left lateral position, an exploratory thoracotomy was performed through the bed of the fifth rib. Exploration of the mediastinum revealed a firm, smooth, oval-shaped tumor mass present just above the left main pulmonary artery and below the aortic arch. Further dissection revealed that the left vagus nerve entered the tumor mass and emerged from it. The recurrent laryngeal nerve arose approximately 5 mm. above the tumor. At the distal end of the tumor was the remaining normal trunk of the vagus nerve and the pulmonary plexus. The tumor was removed amputating the vagus nerve superiorly just below the recurrent laryngeal.

The patient tolerated the procedure well, and no evidence of rise of pulse rate or drop in blood pressure was seen at any time. There was no evidence of laryngeal paralysis. Postoperative examination of the larynx revealed both cords of equal amplitude.

Pathology Report.—Gross Examination: The specimen was a piece of tissue 5 by 2 by 2 cm. which was pink, smooth, and at one point, contained a firm 0.5 cm. nodule. The tumor appeared cystic. On cut surface the specimen was gelatinous.

Microscopic Examination: Neurofibroma of the vagus nerve.

The patient had an uneventful postoperative course and was discharged from the hospital on Jan. 8, 1954.

SUMMARY

A case of neurofibroma of vagus nerve in the chest unassociated with generalized neurofibromatosis is presented. The tumor was successfully removed.

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VENTRICULAR FIBRILLATION IN ARTIFICIAL HYPOTHERMIA

J. García Ramos, M.D.
Tlalpan, D.F., Mexico

ARTIFICIAL hypothermia is now increasingly attracting the attention of physicians and surgeons for its many applications in several fields. In its use as a valuable tool in cardiac surgery, however, the surgeon has been faced with the problem of ventricular fibrillation that seems to occur more readily than at normal temperatures. Bailey and associates[1] have reported ventricular fibrillation as occurring in 66 per cent of their cases; other authors have similar figures.[2]

There is not a thorough agreement on the mechanism of ventricular fibrillation. There are, however, some well-established facts. One condition for the appearance of fibrillation is heterogenicity in the properties of the ventricular muscle.[3, 4] Another condition is the initiation of an impulse at the end of the functional refractory period of the preceding one.[4, 7] It was decided, therefore, to apply these known facts to the experimental study of this cardiac disorder under hypothermia with the idea of finding some practical applications which might help in solving this important problem.

METHOD

Rats, under Nembutal anesthesia (50 mg. per kilogram, intraperitoneally), were used. A tracheal cannula was inserted and artificial respiration instituted, the chest was opened through removal of the sternum and the costal cartilages, leaving the heart widely exposed. The opened pericardial sac was sutured to the skin. On the ventral surface of the ventricle were placed electrodes for stimulation and recording (small steel serrefines fixed to the epicardium and hanging from fine flexible wires). The animal was then placed in a small plastic box, closed except for some openings for the artificial respiration tube, the electrodes, and the thermometer (which was a large cuvette mercury type placed inside the abdominal cavity through a small opening in the anterior wall).

The properties of the ventricular muscle studied were: (*a*) the threshold voltage to electrical stimulation with square pulses of 1 millisecond duration and at a frequency just above the sinusal rhythm; (*b*) the functional refractory period, for example, the shortest interval between two propagated responses to two shocks of 150 per cent threshold voltage, or the maximal frequency at which the ventricle could be driven with a short train of pulses of that magnitude; (*c*) the conduction velocity of the impulses, judged by the latencies for shocks of that same intensity and of frequency just above the actual heart

From the Physiology Department of the Instituto Nacional de Neumología, Tlalpan, D.F., Mexico.
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rate; (*d*) the voltage amplitude of the electrical responses recorded from any point on the surface of the ventricle to an indifferent electrode placed on the neck of the animal and grounded; and (*e*) the rate of rise of the ascending limb of the monophasic action potential.

Once the basal values of these properties were obtained, the box was surrounded by cracked ice and several measurements of those properties were

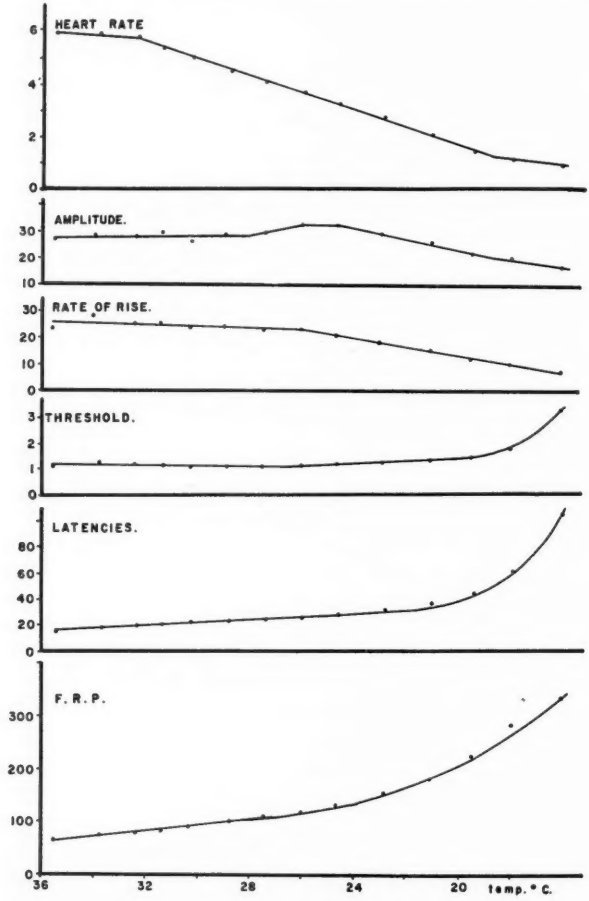

Fig. 1.—Curves from one experiment showing the changes in some ventricular properties during gradual cooling. Ordinates from above to below: Heart rate, in beats per second. Amplitude of the monophasic action potential in millivolts. Rate of rise of the ascending limb of the monophasic action potential, arbitrary units. Threshold to electrical stimulation, in volts. Latencies of the stimulated responses, in milliseconds. Functional refractory period, in milliseconds. Common abscissas: Temperature in degrees centigrade.

made during the cooling period. The records were photographed from a cathode ray oscillograph (DuMont 322) using their own D.C. coupled amplifier. In some cases the records were taken on a three-channel inkwriter galvanometer (Model III D Grass electroencephalograph). In this last case, the action potentials were fed through a Grass Converter-Demodulator and re-

corded with a low frequency galvanometer coil in order to have accurate records due to the infinite time constant of this circuit. The stimuli applied came from a model S4B Grass stimulator through a Grass stimulus isolation unit.

RESULTS

A. *Normal Beats.*—The heart rate changes linearly with the temperature. The thermal coefficient (Q_{10}) has an average value of 2.39, the lowest figure found was 1.77 and the highest one was 3.07.

The voltage amplitude of the action potential practically does not change until a critical point, about 25° C. Below this value, the action potential begins to decline rapidly. Sometimes before that point in the cooling process is reached there is a gradual increase of the amplitude which can be as much as 10 to 20 per cent of the control values.

The changes in the rate of rise of the ascending limb of the monophasic action potential follows a parallel course with the changes in amplitude (Fig. 1).

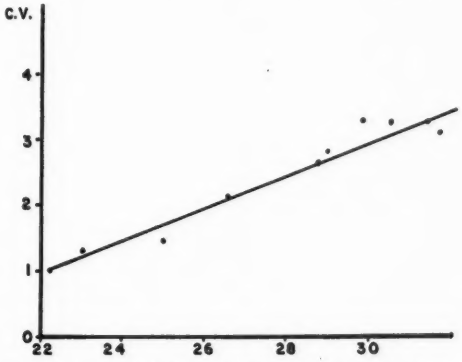

Fig. 2.—Relation between conduction velocity of impulses (reciprocals of the latencies in milliseconds × 10^2, ordinates) and rate of rise of the ascending limb of the monophasic action potential (arbitrary units, abscissas).

B. *Responses to One or Two Stimuli.*—The threshold voltage either does not change at first or presents a gradual decrease of small magnitude until a temperature of about 20° C., at which it tends to rise rapidly (Fig. 1).

The latencies of the responses increase in an exponential-like manner. The changes are very small for temperatures above 25° C., usually not reaching values greater than 25 per cent of the control even at this particular temperature. After that point a slight reduction in temperature results in a great increase in latency (Fig. 1).

There is a linear relation between conduction velocity of impulses and the rate of rise of the ascending limb of the action potential (Fig. 2).

The functional refractory period follows a parallel change, although the increase is always greater than the corresponding variation in latency. At 25° C., it is usual to record an increase of 50 per cent, and sometimes even 100 per cent above the control values.

C. *Responses to Trains of Stimuli.*—The responses to trains of stimuli, of 1 second duration, at different frequencies, and of intensity of 150 to 200 per

cent threshold, were recorded. Successive latencies and amplitudes of the responses were measured.

During the control conditions the amplitude of the responses are more or less the same for the different frequencies of stimulation. At higher frequencies (12 to 16 per second), there is a gradual fall in amplitude for the successive responses until suddenly the ventricle begins to alternate to the shocks. The latencies shortened in the successive responses to relatively low frequencies and showed a moderate increase with the higher ones. With the highest ones, there is some alternation until finally an impulse is missing (Fig. 4).

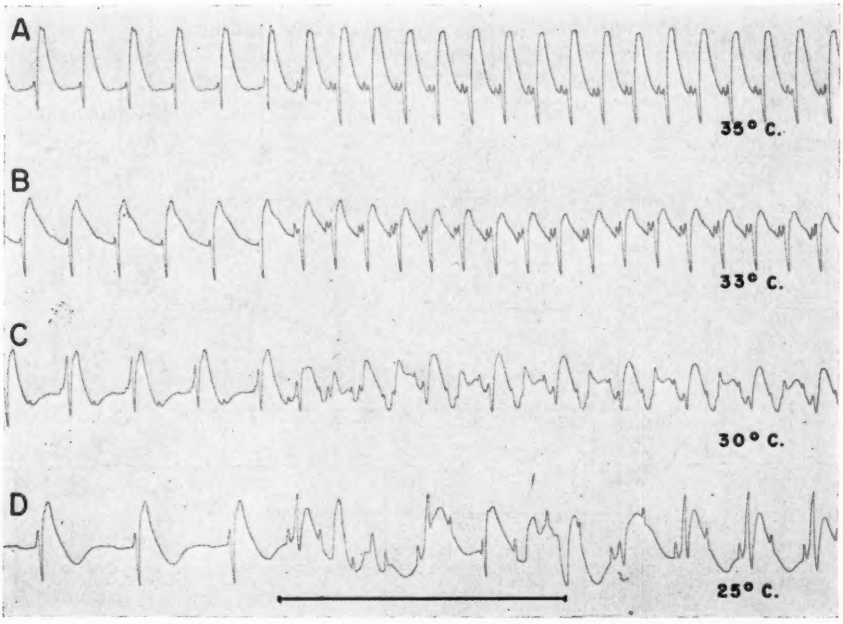

Fig. 3.—Gradual development of heterogenicity in the ventricular muscle with falling of the temperature. The irregularities in the responses show that speed of conduction and refractoriness are not the same for all active fibers. Electrograms taken with the electroencephalograph; stimulating and recording electrodes over the ventricle. Frequency of stimulation 9 per second; the horizontal line below represents one second.

As the cooling advances the amplitudes become irregular, the more so the lower the temperature. Fig. 3 gives some examples for a frequency of stimulation of 9 per second. The latencies increase markedly and show the alternation sooner, as illustrated in Fig. 4, for a frequency of 8 per second. The action potentials for the longer latencies are the smaller ones. Sometimes the action potential is so small that it looks just like an irregular base line.

D. *The Effect of Aconitine.*—Aconitine crystals applied locally over the surface of the ventricle do not give rise to the development of an ectopic foci of impulses at the lower temperatures, even if the heart is made slower by cutting the artificial respiration, or after a train of electrical shocks at relatively high frequency, conditions which always make this kind of ectopic foci to discharge in a heart at normal temperature.

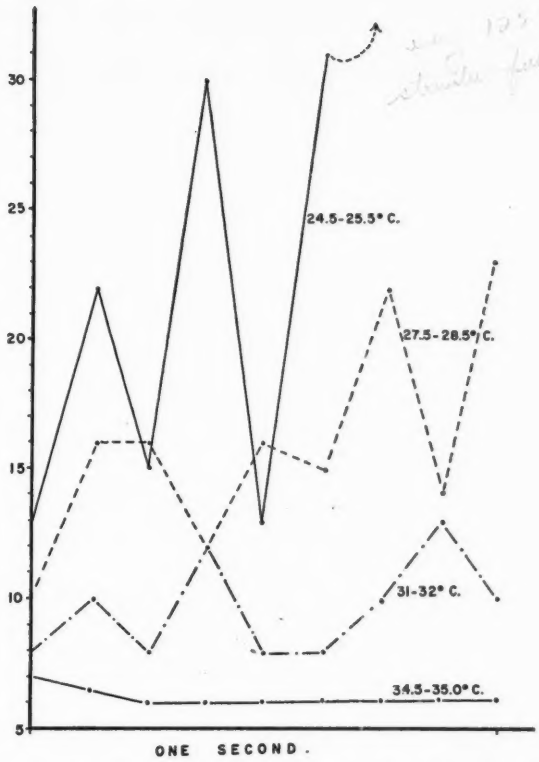

Fig. 4.—Latencies for the individual responses to each of a train of electrical shocks at a frequency of 8 per second and at the temperatures given for every curve. Ordinates: latencies in milliseconds. Abscissas: time (each point is plotted at ⅛ second interval).

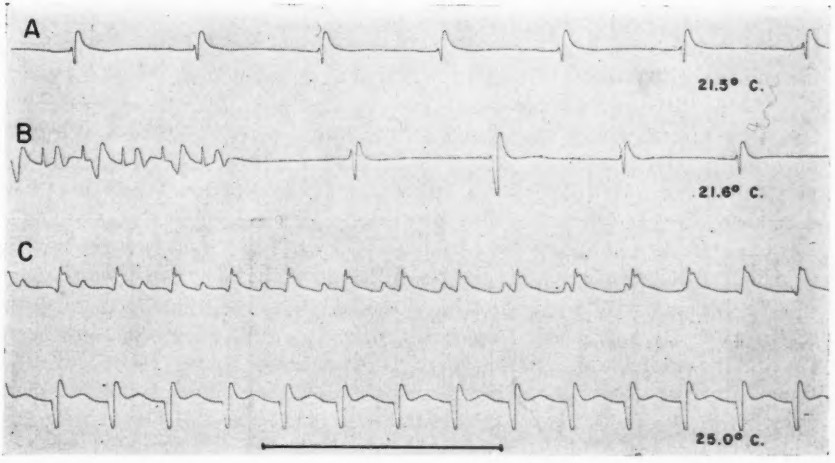

Fig. 5.—Effect of aconitine locally applied on a cool ventricle. Electrograms of the heart: *A*, Normal ventricular beats, in spite of the fact that the aconitine have been already applied. *B*, After starting the warming up. There is some extrasystolic activity but only after a train of electrical shocks at a frequency of 6 per second. *C*, At a higher temperature an ectopic focus begins to discharge. Here, the upper record shows the auricular activity at a lower rate and was taken from an electrode on the right appendix to another point on the ventricle. Time calibration: one second.

When the animal is warmed again, the aconitine applied may have the usual effect (Fig. 5).

DISCUSSION

Some of the results can be analyzed from the pure physiologic point of view. The amplitude of the monophasic action potential, the rate of rise of the ascending limb, and the threshold to electrical stimulation vary in a manner similar to that described for the nerve fibers.[5, 6]

For the increase in height of the action potential, no satisfactory explanation has been advanced. For the decline of those properties it is interesting to recall that Lorente de No[6] pointed out that "the effect of a lowering of the temperature closely resembles that which is produced by lack of oxygen," and that "the changes in the spike potential are referable mainly to a reduction of the rate of those chemical reactions which underlie the alteration itself and the restoration of the membrane potential."

The relation between speed of conduction and rate of rise of the ascending limb of the monophasic action potential is in agreement with the observations of Gasser[5] on the nerve fibers, and also with the local circuit theory for the propagation of impulses.

Let us now examine the data in regard to the mechanism of ventricular fibrillation. According to the circus movement theory to explain this cardiac disorder there is required a closed path of such an extension as to contain an impulse. The wave length of this impulse must then be smaller than the path itself. The heart of the large animals always has the proper dimensions to enclose one or several of those closed paths. Any condition which can shorten the wave length, for example, increasing the speed of conduction, or shortening of the refractory period, or both, will favor the appearance of fibrillation. The enlargement of the possible paths to a traveling impulse will act in a similar way, as does the establishment of heterogenicity in the properties of the individual muscle fibers. All that is necessary in such conditions is to initiate a unidirectional wave of impulse by applying a stimulus at the end of the functional refractory period of a preceding one.[7]

When this study was started it was expected to find, with the lowering of the temperature, a decrease in speed of conduction with either no change in duration of the refractory period or at least with a small increase in this period, but not in proportion with the change in conduction velocity. As the data show, the change in the refractory period, as this is currently determined, was greater than the increase in latency. This result would be against the development of fibrillation, as the wave length of the impulses is now longer.

The results with trains of stimuli showed, however, that latencies do increase markedly at successive responses (Fig. 4). Still more, the heterogenicity in the individual muscle fibers become more evident the lower the temperature is (Fig. 3). This means that the wave length progressively shortens at the same time that the path enlarges by making itself irregular. Thus, the present results are compatible with the circus movement theory as the mechanism for ventricular fibrillation. Even if the refractory period will have shown an increase much greater in proportion to the degree in which conduction velocity was slowed, the simple development of heterogenicity in the muscle fibers alone, as revealed for the variability in latencies and the

appearance of irregularities in the amplitude and configuration of the responses, will be enough to make the ventricle apt to develop fibrillation and of a less easily reversible kind, as is the one found in the clinical observations.

The fact that the threshold of stimulation does not change markedly with the lowering of temperature makes reasonable the possibility of the initiation of an impulse which, under proper conditions, can induce fibrillation.

This type of result shows that sometimes it is not enough to make measurements at static conditions. The two-shock method for determining the length of the refractory period and the latency to the first shock were giving us a wrong picture. The observations with trains of shocks, fortunately, gave us a definite answer.

The studies made by Sherf[8] with aconitine led him to believe that multiple ectopic foci were responsible for ventricular fibrillation; the smaller waves seen in the records taken from a fibrillating heart would be local nonpropagated responses. García Ramos and Miledi[4] have shown, however, in a study with micro-electrodes, that there are no such local potentials, every action potential recorded is a propagated one, and the frequency of activity is just limited by the functional refractory period of the individual muscle fibers at that moment. The irregularities in the records are due to the lack of synchronization of the different active elements.

The present observations with aconitine are not a direct disproof of the multiple foci theory. In fact, the effects of this drug cannot be used in support of any theory. The conditions of its action are artificial. The only thing one can say is that the lack of effect of aconitine at lower temperatures, which was described precisely by Sherf,[8] makes all the arguments based on its action unsuitable for the explanation of the mechanism of ventricular fibrillation.

On the other hand, there is not any evidence that the potential automatic activity of the ventricular muscle is as good at low temperature as it is at the normal one.

The clinical observations of Bigelow and co-workers[2] that maintenance of blood pressure above 80 mm. Hg and hyperventilation of the patients give some protection against the ventricular fibrillation of hypothermia suggest that hypoxia of the muscle can be a factor. It is true that at low temperatures the oxygen consumption of the heart is greatly diminished and that the fall in blood pressure could be not serious enough to interfere with the proper irrigation of the muscle. If one takes into account, however, that aortic pressure is not the only factor in coronary circulation and that the energy of the contractions and the heart rate have also a direct effect on improving it, as shown by García Ramos, Alanís, and Rosenblueth[9] in the isolated, perfused dog's heart, we have a greater support for this idea. In clinical as well as in the experimental observations, it is found that the heart rate decreases markedly, and Bigelow and co-workers[2] found that the cardiac output shows a progressive fall during cooling.

It was pointed out previously that the changes in the electrical events of heart activity can possibly be ascribed to a failure of the oxygen utilization mechanisms.

There is another observation that favors the idea of hypoxia of the heart muscle at low temperatures. Swan[10] has reported that acetylcholine and Prostigmin seems to protect the heart against fibrillation, and García Ramos and Ramírez de Arellano[11] showed that acetylcholine depresses the oxygen consumption of frog's and turtle's hearts.

An analysis of the conditions for establishment of heterogenicity in the properties of ventricular muscle fibers due to hypoxia permits one to state some practical measures as follows: (*a*) try to maintain the coronary blood pressure as high as possible; (*b*) keep the arterial oxygen saturation at normal values; and (*c*) reduce the sympathetic activity, the local as well as the general. Adrenalin is now a well-known hypoxiating agent.[12] The same can be said about some other sympathomimetic amines. All these measures will tend to reduce the heterogenicity of the heart muscle and so give some protection against fibrillation.

SUMMARY

An experimental study on rats under artificial hypothermia shows that lowering the temperature of the animal increases the heterogenicity of the ventricular muscle making it more apt to present ventricular fibrillation. This change is probably produced by the depression of oxidative reactions and by the reduction in the proper irrigation of the heart. These two factors together interfere with the processes of restoration after activity.

A discussion is made on the physiologic significance of the changes with respect to the mechanism of ventricular fibrillation. The data are in favor of the idea of a circus movement more than that of multiple ectopic foci.

Some practical applications are derived from the analysis of the results and of the clinical observations. Reduction of extra oxygen consumption and an adequate oxygenation of the heart muscle will tend to protect it against fibrillation.

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THE ACUTE EFFECTS OF RESECTION OF PULMONARY TISSUE ON SOME PULMONARY FUNCTIONS IN THE DOG

M. Henry Williams, Jr., M.D.,* Philip C. Canney, Captain, MC, USA, and Claudia R. Rayford, Ph.D.
Washington, D. C.

EVALUATION of the acute response of anesthetized dogs to resection of pulmonary tissue and to reduction of the blood supply to the lung has been confined largely to hemodynamic studies. Long and associates,[1] found that pneumonectomy caused a 2 to 13 mm. water rise of the mean pressure in the right ventricle and a variable effect on the cardiac output. Fineberg and Wiggers[2] found that failure of the right ventricle developed only after approximately 60 per cent of the pulmonary artery was occluded. Gibbon and Churchill[3] found that occlusion of approximately 60 per cent of the cross-sectional area of the pulmonary artery produced a fall of systemic blood pressure whereas the systemic pressure remained constant or was elevated after occlusion of from 40 to 73 per cent of the lobar branches of the pulmonary artery.

This report concerns the immediate effects of resection of the left lung and of the upper and middle lobes of the right lung on alveolar and arterial blood gas tensions, the mean pressure in the pulmonary artery and the systemic arteriovenous oxygen difference in 6 dogs. These data were obtained in order to assess the effect of resection of large amounts of pulmonary tissue on various aspects of pulmonary function of the remaining lung and to obtain information as to the minimum amount of pulmonary tissue that must be preserved in order to carry on these functions.

METHODS

Dogs were anesthetized by the intravenous injection of Pentothal (25 mm. per kilogram) followed by barbital sodium (200 mg. per kilogram). The dogs were ventilated through a tracheotomy tube by means of a constant-volume Bird respirator pump. This pump permitted collection of the expired air and substitution of a low-oxygen mixture for room air as the inspiratory gas. Pump rate and stroke volume were kept constant throughout the experimental period. The lungs were kept inflated throughout the experiment by passing the expired air through a column of 5 cm. of water. The femoral artery was cannulated for the collection of arterial blood samples. The chest was opened in the fourth to the fifth intercostal space and the sternum was transected. A polyethylene

From the Department of Cardiorespiratory Diseases, Army Medical Service Graduate School, and Anesthesia and Operative Service, Walter Reed Army Hospital of Walter Reed Army Medical Center, Washington, D.C.

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*Present address: Cardiorespiratory Centre, Grasslands Hospital, Valhalla, N. Y.

cannula was inserted through the wall of the right ventricle (through a 17-gauge needle) into the pulmonary artery for measurement of the mean pressure in the pulmonary artery and for collection of mixed venous blood. The pressure at the level of the catheter tip was measured at the end of each experiment and was used as the zero pressure. After a 20-minute period of ventilation with room air, expired air was collected for three minutes. Samples of arterial and of mixed venous blood were drawn simultaneously at a constant rate throughout this 3-minute period. Twelve per cent oxygen was then administered as the inspiratory gas and, after twenty minutes, the air and blood samples were collected again. Ventilation was then re-established with room air, and the roots of the left lung, right upper and middle lobes were ligated and clamped and the pulmonary tissue distal to the clamps was resected. After a 20-minute interval, expired air and blood samples were obtained and the dog was placed on 12 per cent oxygen. Twenty minutes later the final expired air and blood samples were collected.

Arterial blood gas tensions were measured immediately by the direct technique developed by Riley and his co-workers.[4] The equilibrating water bath was kept 0.4° C. lower than the dog's rectal temperature, approximately the temperature existing in the lung.[5] Expired gas volumes were measured in a Tissot spirometer and corrected to 38° C. [BTPS (body temperature pressure, saturated)] for calculation of ventilatory volumes and to STPD (standard temperature pressure, dry) for calculation of oxygen consumption. Expired gas composition was measured on the Scholander microgas analyser[6] and the respiratory quotient was calculated. The alveolar oxygen tension was calculated from the alveolar equation,[7] substituting arterial for alveolar carbon dioxide tension. The lung dead space was calculated from the Bohr equation. The mean pressure in the pulmonary artery was measured on a saline manometer. The arterial and venous oxygen saturations were measured by a spectrophotometric technique.[8]

RESULTS

Data obtained from the 6 of 7 animals which survived throughout the experimental period are presented in Table I. In each animal the arterial carbon dioxide tension, both during the inspiration of room air and of 12 per cent oxygen, was lower after resection of the pulmonary tissue than it had been during the control period. Since the expired carbon dioxide tension was virtually unaffected by pneumonectomy the lung dead space was always lower after resection [83 to 95 per cent (average: 86 per cent) of the control during room-air breathing and 82 to 94 per cent (average: 88 per cent) during 12 per cent oxygen breathing]. The arterial oxygen tension measured during inspiration of room air increased in 4 of the dogs after resection. In all but 1 animal the alveolar oxygen tension rose. The alveolar-arterial oxygen tension difference did not change significantly. During the inspiration of 12 per cent oxygen the arterial oxygen tension was higher in 5 of the animals after resection, the alveolar oxygen tension was higher in all of the dogs, and the alveolar-arterial oxygen tension difference was not altered significantly.

TABLE I

DOG NUMBER	INSPIRED OXYGEN TENSION (MM. HG) $P_{I_{O_2}}$	EXPIRED CARBON DIOXIDE TENSION (MM. HG) $P_{E_{CO_2}}$	RESPIRATORY QUOTIENT R	ARTERIAL CARBON DIOXIDE TENSION (MM. HG) Pa_{CO_2}	ARTERIAL OXYGEN TENSION (MM. HG) Pa_{O_2}	ARTERIAL OXYGEN SATURATION (PER CENT) Sa_{O_2}	VENOUS OXYGEN SATURATION (PER CENT) $S_{\bar{v}O_2}$	HEMATOCRIT HCT.	DEAD SPACE C.C. (BTPS) V_D	OXYGEN CONSUMPTION C.C./MIN. (STPD) V_{O_2}	PULMONARY ARTERY PRESSURE (CM. SALINE) PAP	EFFECTIVE ALVEOLAR OXYGEN TENSION (MM. HG) $P^e_{A_{O_2}}$
1	145	10.3	0.81	33	87	93	70	48	98	86	22	106
	85	8.5	0.94	31	51	80	42	48	103	63	21	52
	145	10.7	0.87	31	82	92	51	53	84	82	29	110
	85	8.8	1.03	30	56	82	44	54	95	56	27	56
2	148	13.1	0.80	53	87	92	64	57	64	79	27	84
	87	13.2	0.85	57	34	48	28	60	66	75	36	22
	148	12.9	0.80	47	85	92	62	61	55	75	39	92
	87	12.9	0.91	56	29	37	20	64	62	66	51	25
3	147	10.1	0.76	35	95	90	58	37	127	104	18	103
	86	9.9	0.84	36	50	86	40	40	130	93	30	44
	147	11.1	0.78	31	96	93	49	44	105	108	27	109
	86	10.0	0.88	31	55	80	40	44	115	88	30	51
4	148	11.8	0.70	60	70	75	67	52	63	76	29	68
	87	8.7	0.87	66	16	15	10	57	72	45	40	12
	148	12.6	0.75	58	80	83	76	53	53	71	37	75
	87	9.8	0.97	58	28	20	11	53	61	42	44	27
5	149	12.5	0.86	42	90	92	45	36	76	86	21	102
	87	13.2	0.93	43	37	39	11	38	80	84	36	41
	149	12.6	0.85	41	96	85	25	38	63	95	33	102
	87	12.6	0.98	41	40	35	6	35	64	83	47	45
6	149	15.3	0.81	51	84	85	73	46	64	99	30	89
	87	14.2	0.93	48	38	49	35	46	65	80	31	36
	149	13.0	0.84	44	91	90	61	44	61	79	38	98
	87	13.2	1.05	38	55	76	50	45	53	64	39	51

Data on 6 dogs, before and after resection of the left lung and the right upper and middle lobes of the right lung. In each dog, the first two lines represent control observations, first during inspiration of room air, then of 12 per cent oxygen, and the second two lines are the data obtained after resection, first during inspiration of room air, then of 12 per cent oxygen.

During room-air breathing the mean pressure in the pulmonary artery was always higher (7 to 12 cm. saline) twenty minutes after resection of lung tissue than it had been during the control period. The oxygen consumption was approximately unchanged before and after resection in 5 of the dogs, and in Dog 6 it fell. Since the arterial-venous oxygen difference was larger after resection in all but 1 dog, the cardiac output was diminished (33 to 89 per cent) in all except one experiment. The calculated pulmonary vascular resistance (pulmonary artery pressure/cardiac output) was increased in each instance (120 to 385 per cent of the control, average: 215 per cent). During the inspiration of 12 per cent oxygen the pressure in the pulmonary artery was, with one exception, elevated after resection and the calculated resistance to flow through the lung was always increased.

DISCUSSION

These experiments concerned the effects of resection of a large percentage of the lung (according to data of Rahn and Ross,[9] approximately 68 per cent of the total lung). Six dogs survived the procedure, and the seventh dog died two minutes later. All the surviving dogs showed no impairment of the pulmonary functions studied. Although the pressure in the pulmonary artery increased, cardiac output was sufficient to maintain the oxygen consumption at the control level in 5 of the dogs. Since pulmonary ventilation remained constant, and since the dead space was decreased after resection, the arterial carbon dioxide tension fell while the alveolar oxygen tension rose. The alveolar-arterial oxygen tension difference was not significantly increased, except for Dog 1 in which the tension difference during room-air breathing increased 9 mm. Hg. In the other dogs, the changes were within the limits of experimental error (7 mm. Hg). Since this tension difference is a reflection of different phenomena during the inspiration of room air and during the inspiration of a low-oxygen mixture, two inferences may be drawn from these data. The tension difference during room-air breathing is due to the presence of venous admixture to arterialized blood and to the presence of different ventilation-perfusion ratios in different parts of the lung.[10, 11] Since the tension difference did not increase after resection of 68 per cent of the pulmonary tissue, pulmonary ventilation–perfusion relationships in the remaining lung were apparently unchanged. The absence of a significant tension difference during low oxygen breathing after resection of 68 per cent of the pulmonary tissue indicates that the diffusing capacity of the lung was so high that no measurable difference existed between the oxygen tension in the alveoli and in the blood leaving the alveoli. In these dogs, the average arterial carbon dioxide tension after resection, during 12 per cent oxygen breathing, was 44 mm. Hg and the average effective alveolar oxygen tension was 43 mm. Hg, an insignificant difference. Since the average arterial-venous difference was 27 per cent saturation, the difference between the alveolar and the mean capillary oxygen tension was 8 mm. Hg or less.[12] The average oxygen consumption was 67 c.c. per minute, so the oxygen diffusing capacity averaged 8 c.c. per minute per

millimeter of mercury or greater. Since only 32 per cent of the lung tissue was present, the diffusing capacity of the entire lung would have been at least 25 c.c. per minute per millimeter of mercury.

Although resection of pulmonary tissue seemed to have no discernible effect on pulmonary blood-gas relationships, there was an obvious increase in the pulmonary vascular resistance. From the data in Table I the resistance during room-air breathing increased to 120 to 385 per cent of the control (average: 217 per cent) and during 12 per cent oxygen breathing 129 to 386 per cent (average: 215 per cent). This was not as great as one might expect on mechanical grounds alone when 68 per cent of the vascular bed was removed. A previous study of the perfused lung of the dog has revealed that a high intravascular pressure serves to increase the size of the pulmonary vascular bed and thereby decrease pulmonary vascular resistance.[13] Thus, the calculated resistance is a function of the pressure in the pulmonary artery, and one cannot relate the resistance to the amount of lung tissue present unless the pressure remains constant.

The effect of further reduction of the pulmonary vascular bed was investigated in 5 of these dogs. In each instance, ligation of the right lower lobe, leaving approximately 8 per cent of the lung in place,[9] resulted in extreme pulmonary hypertension (59 to 88 cm. saline), followed, in five to thirty-five minutes, by a fall of the pressure in the pulmonary artery, dilatation of the right ventricle, and death. In 4 of these animals marked arterial unsaturation developed during the period of pulmonary hypertension (25 to 36 per cent in 4 dogs, 88 per cent in the fifth) and was completely overcome, in the two instances studied, by the inspiration of oxygen. Such a marked effect of oxygen indicates that the reduction of the arterial oxygen saturation was largely due to a diminished diffusing capacity of the lung so that a large difference existed between the oxygen tension in the alveoli and in the blood leaving the alveoli. Inspiration of oxygen raised the alveolar oxygen tension to some 700 mm. Hg and increased the rate of diffusion of oxygen across the alveolar membrane into the blood. A similar phenomenon occurs after diffuse embolization of the lung.[14] The reduction of diffusing capacity after resection of 92 per cent of the lung may be attributed to the removal of diffusion surface and to reduction of alveolar perfusion of the remaining lung resulting from overdistention of the alveoli by the undiminished ventilatory volume which was necessary for maintenance of adequate alveolar ventilation. The close relationship between the circulatory and diffusion functions of the lung is indicated by the fact that both were relatively unimpaired after resection of 68 per cent of the pulmonary tissue whereas right heart failure and limitation of diffusion of oxygen both became apparent after resection of 92 per cent of the lung tissue. The pulmonary vascular reserve was sufficient to maintain adequate circulation and diffusion of oxygen when 68 per cent of the lung was removed, but 8 per cent of the lung contained insufficient vascular bed to maintain either of these functions.

SUMMARY

Acute resection of approximately 68 per cent of the pulmonary tissue has been performed in 7 dogs. One dog died. The remaining dogs showed an increase of the pressure in the pulmonary artery, a small decrease in the arterial carbon dioxide tension and lung dead space, no significant change in the alveolar-arterial oxygen tension difference both breathing room air and 12 per cent oxygen, no consistent change in oxygen consumption and an increase in the arterial-venous oxygen difference. The calculated cardiac output fell after this procedure, and the pulmonary vascular resistance rose. Although the resection had a detectable effect on the cardiac output and on the pressure in the pulmonary artery, there was no adverse effect on pulmonary ventilation–perfusion relationships, and the diffusing capacity was so high that, even during hypoxia, there was no appreciable difference between the partial pressure of oxygen in the alveoli and in the blood leaving the alveoli.

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A SIMPLE DEFIBRILLATOR ELECTRODE

SIGMUND A. WESOLOWSKI, CAPTAIN, MC, USA, EDWIN GORDY, CAPTAIN, MC, USA, AND DAVID COLE
WASHINGTON, D. C., AND BROOKLYN, N. Y.

THE authors have designed and constructed a simple and convenient defibrillator electrode for use with a modified Mackay defibrillator[1] (Fig. 1). The electrode is made from a stainless steel ladling spoon, a 10-inch length of ⅜ inch OD stainless steel tubing, and a male banana plug. The handle is severed from a spoon, the stub of the spoon handle is silver soldered into one end of the stainless steel tube. A male banana plug is placed into the other

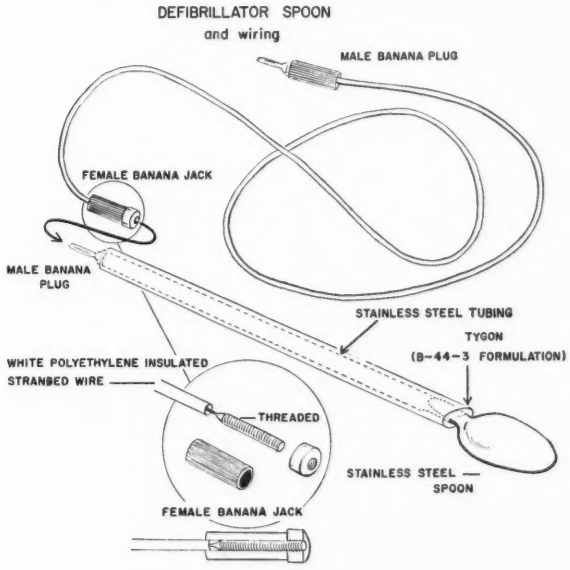

Fig. 1.

end of the rod and silver soldered into place. The back of the spoon is sand-blasted and covered with an insulating coat of baked-on epoxy-resin. The stainless steel rod is insulated by Tygon tubing (formulation B-44-3) of ⅜ inch ID and ⅝ inch OD. This construction permits repeated autoclaving. The

From Division of Surgery, Army Medical Service Graduate School, WRAMC, Washington, D. C., and Department of Surgery, State University of New York, School of Medicine at N.Y.C., Brooklyn, N. Y.

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electrode is connected to the defibrillator by appropriate wiring using polyethylene-insulated stranded wire, which can be boiled, or Teflon-insulated wire, which can be autoclaved. Banana connectors are used at both ends of the leads. The advantages of this electrode are:

1. It is easily constructed and easy to keep clean. It can be autoclaved.
2. The spoon is properly cupped to make good contact with the heart. It gives sufficient area of contact so that there is minimal danger of excessive current density. The danger of burning the myocardium is thereby minimized.
3. The use of banana jacks allows for rapid setup and use.
4. The back of the spoon is insulated to minimize stray currents.

This design of defibrillator electrodes has been successfully used in experimentation involving use of a mechanical heart.[2]

These electrodes supplied by D. Cole, Glenn Echo, Maryland.

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OCCURRENCE OF TUBERCULOSIS IN AN ACCESSORY PULMONARY SEGMENT

J. Rodriguez Arroyo, M.D. (by invitation), S. E. Domm, M.D., D. H. Waterman, M.D., and W. K. Rogers, M.D. (by invitation)
Knoxville, Tenn.

BOYDEN[1] has described anomalous pulmonary segments. Search of the literature has not revealed report of occurrence of tuberculosis in such segments.

The occurrence of tuberculosis in an accessory pulmonary segment of Boyden would therefore seem of sufficient interest to report.

CASE REPORT

Case No. 636.—Twenty-year-old Negro woman.

Diagnosis.—Pulmonary tuberculosis, far-advanced, chronic; original massive bilateral cavitary disease (Fig. 1) with incomplete resolution and assumed inspissation following adequate medical treatment consisting of antituberculosis drugs and bed rest.

In the course of the left-sided resection the distribution of the tuberculosis disease in the upper lobe was considered unusual. The posterior segment was destroyed, as anticipated. There was, however, a wedge-shaped area of contracted, nodular tissue located between the apical segment and the anterior segment (Fig. 2). The posterior segment was uneventfully resected. However, on further dissection in the interlobar fissure, a tubular structure was encountered superficial to the pulmonary artery, and was demonstrated to contain air rather than blood. It was identified as an anomalous bronchus supplying the triangular area previously described. The arterial supply of this area was by a small branch of the anterior segmental artery. The venous drainage was by way of intersegmental veins. The area was identified as a BX^2a subsegment of Boyden (Fig. 3), and was uneventfully resected (Fig. 4).

The case is presented in view of its practical interest, that is, the importance of alertness on the part of the surgeon with regard to anomalous bronchi especially in the course of segmental resection lest bronchi and arteries be transsected.

As Boyden has mentioned, if the operator were to follow the usual method of removing the lingular segments, he would sever bronchus BX^2a and its artery, AX^2a, thus compromising the whole region posterior to the anterior

From the East Tennessee Tuberculosis Hospital, P. M. Huggin, M.D., Director, Knoxville, Tenn.
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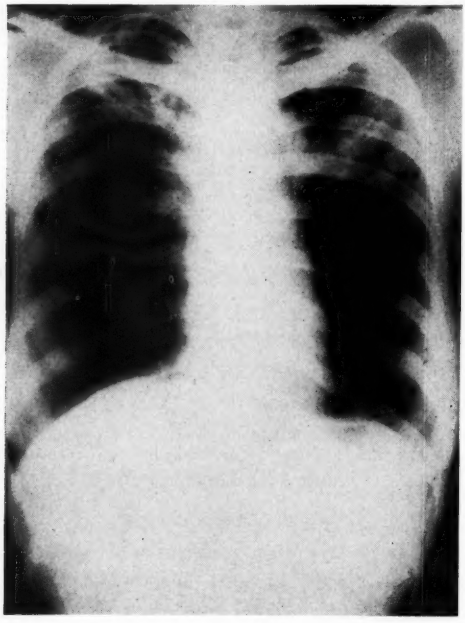

Fig. 1.—Original x-ray.

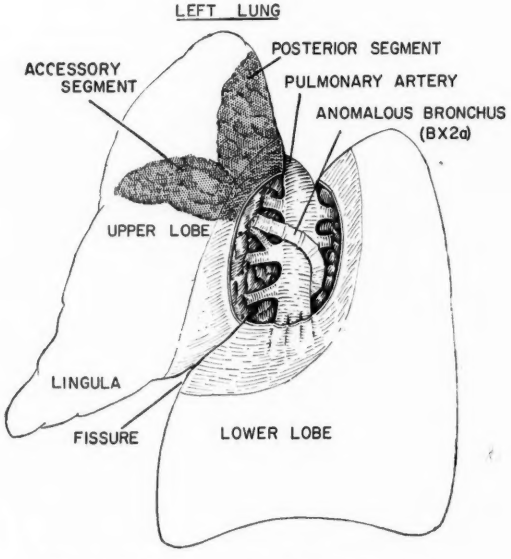

Fig. 2.—Diagram showing findings at operation.

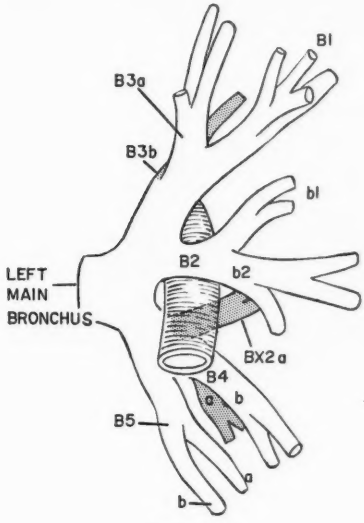

Fig. 3.—Diagram showing accessory bronchus BX^2a according to Boyden.

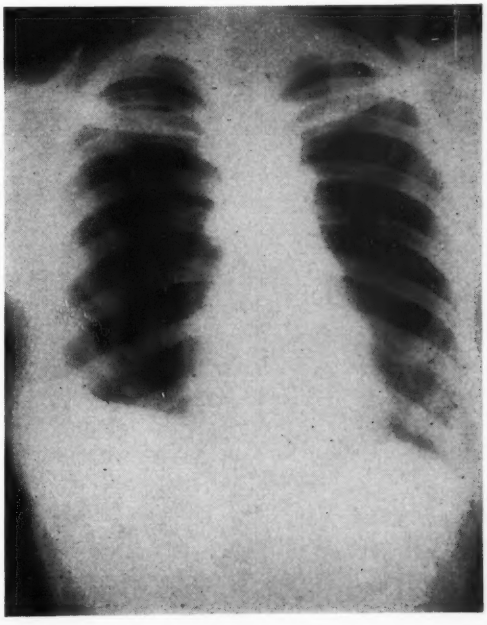

Fig. 4.—Final x-ray following bilateral pulmonary resection.

segment (B^2). Boyden therefore emphasizes the necessity of examining the interlobar surface of the upper lobe before resecting the lingular segments.

The case was of theoretical interest in view of the demonstration of segmental localization of tuberculous involvement, and also in view of the fact that the pulmonary tissues supplied by the aberrant bronchus were involved with infectious disease in spite of the fact that the segments on either side were not significantly involved.

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